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President—Sir D'Arcy Power, K.B.E., F.R.C.S.

Rheumatoid Arthritis: Is it a Deficiency Disease?

By M. J. Rowlands, M.D.

This is a preliminary announcement on a subject so vast that there seems no end to it; and there can be no end to the research connected with it.

It is true that many of my listeners may question the results of my research, but I suggest that the work has brought out certain points which should lead to a discussion beneficial to ourselves as practitioners, both medical and veterinary, and, what is more important, beneficial to mankind as well.

Originally, I had hoped to have read my paper as promised in the next session, when perhaps I should have brought part of the work to an end, but the end I am unable to visualize, therefore I thought the sooner I put my views before this Section the better.

For a number of years I have devoted most of my time both in the consulting room and in the laboratory to the disease to which the name of rheumatoid arthritis has been given. To any medical man who is in the habit of being consulted as I am by sufferers from this fell disease, I felt it was high time that someone took up the investigation. The late Professor Strangeways and myself fought many battles, but the results brought us no nearer the end. Literature helps one but little as there is such a conflict of opinion.

An excellent paper has recently been written by Dr. Tom Hare, of the University of Liverpool, involving enormous work, on rheumatoid arthritis in horses, and published in the two recent numbers of the *Veterinary Record*. I think that Dr. Hare has covered all the work done during the last forty years. He quotes sixty-eight references. Yet after such vast research his conclusion is that: "Bacteriological, histological and cytological examination of the affected fluids and tissues have failed to identify the causal factor."

Dr. Llewellyn, again, makes the following statement in his book on "Rheumatoid Arthritis": "From the foregoing remarks it will be seen that the ætiological problem presented is an extremely difficult one, solution of which is impossible in the present state of our knowledge. While leaning towards the toxæmic theory of its origin, one cannot but be sensible that our knowledge of the more intimate chemical changes involved is very vague, and forbids any pretension on our part to an exact ætiology of the affection."

Some of you must have wondered at the title of my paper; I gave it hours of thought and could give it no better title than one in the form of a question.

My clinical investigations began as far back as 1912, when I installed an X-ray apparatus with the idea of trying to find out what similarity there was in the lesions amongst my cases.

In the war during 1914 and 1915 stationed at Netley. The blood-cultures and joint punctures I carried out proved sterile.

Owing to ill-health I had to relinquish the Service for some time; I returned to it again in 1916 and was given the pathological charge of three hospitals of some 2,000 patients, where I could place as many rheumatoid patients for whom I could find beds, an order being posted in the London area that all true rheumatoids were to be sent to one of my hospitals. In this way I was able to accumulate some 200 rheumatoids and keep them for investigation. But with all this opportunity and all the advantages of able assistance and cordial help for over three years, until May, 1919, nothing of great value was discoverable. In 1916 I wrote a paper which was published in the *Lancet*[1] giving the results of my investigations up to that time.

After the war I again took up the investigation of this disease chiefly owing to my farming instinct. The question of vitamins and the work of Hopkins, Funk, Plimmer and Drummond, was being published. I began to experiment with pigs, as I found that a large number of my pigs which were bred on the open-air system were from time to time suffering from marked stiffness and swollen joints. I began to feed my animals on a full vitamin diet and the result of these experiments was marvellous. There was a complete change in the condition of my herd and I decided to show my experimental animals at the largest Fat Stock Show in

[1] *Lancet*, 1916 (i), 133.

42 Rowlands: *Rheumatoid Arthritis: Is it a Deficiency Disease?*

the world—namely, Smithfield. The result of the first time of showing was every possible prize that I could have won as well as the Cup. This gave me ample proof that in animals' malnutrition lay the seat of investigation. In 1921 I read a paper before the Farmers' Club at the Surveyors' Institute discussing my experiments. Professor T. B. Wood, of Cambridge, and Dr. Crowther, Principal of the Harper Adams College, who opened the discussion, ridiculed all my experiments, and the whole idea of vitamins, and, in fact, the only member of the audience who agreed was Lord Bledisloe. To-day I think both Professor Wood and Dr. Crowther are aware of the value of vitamins and now admit their use to the British farmer. I had already noticed in my patients the frequency of coli bacilluria, and, as I mentioned in 1916, I was convinced that the disease was of trophic origin. My reasons for the theory were the marked wasting of specific muscles such as the vastus internus,—its nerve also supplying the knee-joint; of the deltoid in shoulder cases, the circumflex nerve being involved, also the typical hyperæsthetic areas. I decided to start an investigation on these lines. In January, 1925, I took toll of the *Bacillus coli* examinations of my rheumatoids, and the results of cultures made from the urines of these patients showed that a very high percentage were suffering from coli bacilluria.

In January of this year (1927), I again made records of the bacteriological examination of the urine of my last hundred rheumatoid cases sent to me. Eighty-nine grew *Bacillus coli*; in six only were streptococci found; one grew a paratyphoid B; in four out of the hundred only was I unable to culture any organism.

> The paratyphoid case was a very chronic one; it had persisted from the South African war in 1901, it was a case in which both hips were involved. This patient has done well and now plays tennis. To compare with this case, I mention that of a patient who grew streptococci in his urine and it was found by transillumination that the maxillary antrum was full of pus. He is now free from all pain and able to shoot and walk over ploughed ground. I show the radiographs of both these patients as rheumatoids in whom the same joints were affected, but in whom the infection was due to two completely different organisms.

I was encouraged in this work by the results of Colonel McCarrison's researches. It would be impossible, in the short time that I am devoting to this paper, to describe more than a few of the experiments carried out by myself, and by my colleague Dr. Ethel Browning who has rendered me admirable assistance.

The radiograms which I am showing were kindly prepared for me by another colleague, Mr. J. Sumner Moore. Over 500 radiograms have been taken and some 400 animals have been used during the latter part of this investigation.

As mentioned in my paper in the *Lancet* in 1916, the puncturing of joints and cultivation of the fluid proved to be all sterile, so this side of the work was not carried further. I think that Dr. Tom Hare's valuable work agrees with this finding. The method of investigation carried out by me in human beings when a case was sent with rheumatoid arthritis was the following: The history of the patient was taken in the ordinary way as in hospital, a complete physical examination with notes made on muscular wastings, disabilities, hyperæsthetic areas as well as a minute history of any injury to any part of the body. This was rather important as undoubtedly injury was a marked predisposing cause as to in which part of the body the disease began.

The chest and abdomen as well as the thyroid were carefully examined for any abnormality, blood-pressures in the latter cases were also taken as well as the temperatures. Following this, the nose was thoroughly examined, cultures were taken, and the antrum of Highmore and the frontal sinuses were transilluminated. The teeth, if any, were X-rayed; the condition of the gums was noticed, tonsils were examined and cultures taken. If a patient had a cough, sputum was examined—specimens of urine were passed into a sterile bottle through a sterile funnel; the stools were examined for any abnormal organisms and in the case of female patients if any vaginal discharge was present a speculum was passed and cultures taken from the cervical canal as well as from the vagina.

I have here twenty radiographs. I could show you numbers more but I presume that twenty will suffice to prove that the toe-joint is a joint very commonly involved and you will note that the osteophyte in the majority of cases—if not in all—arises from that part of the joint which is most liable to injury in the modern boot. This joint, as you will realize if you give it a moment's thought, is the one that bears most of the strain in every step we take. Many other joints are affected in the same way. Having found that such a high percentage,

namely 89 per cent., of my patients suffered from coli bacilluria, I determined to find how commonly *Bacillus coli* occurred in the so-called normal subject, and I therefore began by experimenting with my pigs.

Of course, it was found impossible to bring these pigs to my London laboratory and I decided, with the cordial assistance of our Medical Officer of Health, to build a slaughter house on the farm; licence for its construction was granted, as it would have been impossible except in a private slaughter house to carry out any bacteriological technique. A pig begins to eat solid food at about three weeks of age and is killed for pork if properly fed at about four months. The pigs were shot with the captive bullet: they were immediately hung up, the bladder removed, and by means of sterile forceps and scissors a window into the bladder was made, some of the urine was removed, put into broth and brought to my laboratory. Not one of the 100 that were taken grew anything but a few staphylococci, not a single *Bacillus coli* was grown. I think this speaks well for our technique. The animals were killed at the rate of twenty per week. I then interviewed the Home Office and asked to be allowed a licence for my farm permitting me to carry out feeding experiments on my pigs with a deficiency diet. This, I was informed, was impossible, and on no account was I to feed a pig with any foods that I considered were harmful to the animal, as no licence could be granted other than to a recognized institution.

I had by me all the notes of an experiment I had carried out a few years previously. Feeding experts were constantly advising farmers—and are doing so to-day—to feed their pigs on by-products from margarine factories, such as palm kernels, coco-nut, earth-nut, soya beans, etc. So I placed three pens of pigs on these foods as a test, using against them a food containing meat, yeast, cod-liver oil and a salt mixture, the carbohydrate content of the diet being the same in all the pens. Within a few weeks it became apparent that the pigs on a diet of palm-kernel and coco-nut were rapidly going downhill; and at the end of the test the pigs fed on my mixture had increased by 143 lb., and for every 1 lb. of increase in weight had consumed 2·62 lb., whereas the "palm kernel pigs" had increased only 40 lb., and for every 1 lb. of increased weight they had consumed 5 lb. The palm kernel pigs showed a vitamin B deficiency. The stools of all the pigs were brought up to the laboratory from time to time, and investigations in fat absorption were carried out as well as in protein expulsion.

This, of course, put an end to the employment of pigs, and it was decided to use the rat for the experiments. A building which had been used as a maternity home for my sows was rapidly converted into an animal house; fifty breeding does were purchased and breeding was immediately started. The rats were fed on 15 per cent. vitamealo, 25 per cent. barley meal, 60 per cent. middlings. This is a proprietary food which I know has a high fat soluble and vitamin B content, and contains as well an excellent protein and salt mixture. This ensured that the rats had a normal diet and that there was no vitamin deficiency. Twice a week some green food was given to the rats, and it was surprising how the fecundity figures rose and how rapid was growth in our animals. So good were the animals that Professor Drummond reported that the younger ones were of no use for any fat soluble vitamin experiments as they were so saturated with it that it took a great deal of time to make them deficient.

The rat is not such an easy animal from which to take urine cultures as is the pig, and at first our cultures were constantly contaminated. But Dr. Ethel Browning suggested, instead of making a median incision to make the incision apron-form and to turn down a huge flap, thus exposing the bladder. The bladder was removed by sterile forceps and sterile scissors, and the whole organ immersed into broth for culture.

Of the last 100 rats (as soon as our technique was corrected) of the vitamin-fed animals, all proved to be sterile on culture. This made me somewhat despondent, and the next point was to try to see how common the presence of *Bacillus coli* was in the normal human subject. I decided that a medical student had quite a good kind of physique and was a healthy type of human being. Professor Plimmer of St. Thomas's Hospital came to my assistance and placed at my disposal the whole of his first- and second-year students. Forty students were tested; the urine was passed into a sterile funnel and then into a sterile bottle, and cultured. Four were found to be suffering from coli bacilluria, i.e., an occurrence of 10 per cent. in the normal man. It was rather interesting to note that in one of these cases the urine contained sugar, in another the appendix was removed a short while after the experiment, another, a third student, was suffering from boils, and the fourth was neurasthenic.

44 Rowlands: *Rheumatoid Arthritis: Is it a Deficiency Disease?*

It was decided to omit further bacteriological inquiries for the present and to investigate, if possible, the incidence of *Bacillus coli* in the urine of my rheumatoid patients, and see whether intestinal toxæmia played any part in the disease, as I found that in a number of my cases there was tenderness in the right iliac fossa. X-ray examination showed a distension of the colon and delayed expulsion of the barium or bismuth employed. I therefore decided to concentrate my attention on the cæcum and colon.

The rat has no appendix, but the chicken has two appendices, in respect of which I hope on some future occasion to show some further findings.

Fifty rats were brought up from the farm to the Bio-chemical Department of University College, and they were fed on a vitamin B deficiency diet. The diet consisted of heated casein, butter, rice-starch and the ordinary salt mixture. Rice-starch 650 grm., butter 100 grm., casein 200 grm., salt mixture 50 grm. The animals were kept in a heated room in cages. They were rats about six months old, and were all males. They had been bred by me on the farm and their diet from birth was known. They were well grown, almost to full size, and were very healthy. Within three weeks on a deficiency diet the animals were showing signs of going downhill. They were losing weight, not eating their usual quantity of food. their coats lost their shine, they became bald and were constantly eating their excreta, and were not playful—in fact, had all the appearance of a sick animal. We waited a further week, but unfortunately a number of the animals died, and those dying during the night in the absence of the assistant were usually eaten by the others; a normally fed animal never does this. The remaining animals, some thirty in number, survived until the fifth week, when their condition was pitiable to see. We killed these by means of chloroform, and a marked loss of the peristaltic wave was most evident. In a number of them there was slight fluid in the pericardium, but the most marked symptom was absence of the peristaltic wave, general malnutrition and distention of the cæcum. Cultures were made from the urine in the ordinary way described above, but were sterile; sections were made from a large number of these. The next point was to compare fifty normal animals with these deficiency animals, and soon fifty rats were killed, all in the same manner with chloroform, and immediately opened, the peristaltic wave which persisted for some time was watched and the pericardium was examined as well. Portions of the cæcum, transverse colon and stomach were removed for histological examination.

Just at this period, Professor Drummond offered for my use some rats which he had kept for thirty-five weeks on a deficiency diet, though not a total deficiency diet, as he had given them a little marmite. I immediately took advantage of this kind offer, and I shall be able to show you with the epidiascope, sections of the cæcum at about the junction of the ileocæcal valve and also the transverse colon. A little later I visited Professor Plimmer at St. Thomas's Hospital and discussed my investigations with him. When I told him of the difficulty I had had in keeping my rats alive long enough on a complete deficiency diet he pointed to a male rat that he had kept for some eighty weeks on a partial deficiency diet; the rat having had 2 per cent. marmite with its other food. He offered me the animal and I immediately took possession of it. Although the rat looked ill, it had not lost weight for months, its respirations were rapid and one could see that only one lung was active. It was killed by chloroform; one lung was found to be solid; the whole of the mediastinum was full of large glands which looked tuberculous but on examination were found to be pneumococcal. There was no peristalsis, but an abnormal amount of abdominal fat. I now decided to bring up another fifty rats from the farm to University College and they were placed on the deficiency diet, to which was added 2½ per cent. marmite for six weeks. At the end of this period the marmite was removed and for three weeks they were on a complete deficiency diet. Four of these rats were fed on a mixed broth emulsion of pneumococci and staphylococci added to milk. The culture was 1,000 million of each organism per 1 c.c. Each rat took 1 c.c. It was decided to keep these rats for four days, but one died before it had an opportunity of taking the mixture, two were killed forty-eight hours after receiving the mixture, and one was killed four days after taking it, by means of chloroform. Complete absence of peristalsis was noted in all these.

It was now decided to feed nine deficiency rats on a streptococcal broth emulsion, added to milk. They had been on the same diet as the pneumococcal rats up to nine weeks, then for three weeks on 2 per cent. marmite, then for four days on complete deficiency diet. The temperatures of these rats were taken, and, as usual (one of the first symptoms of a vitamin B

deficiency diet) the temperature fell enormously, ranging from 85° F. to 96°; the normal being 100°. This I consider is a most important fact and one on which we, as medical men, should lay great stress. I will show you the temperature chart of a patient suffering from rheumatoid arthritis who was in fairly good condition and was quite an early case. It is useless repeating these temperature charts as they are all similar and we know that with a subnormal temperature infection with any organism is common. Another further batch of normal rats was brought up and fed with pneumococci and staphylococci, each receiving 1 c.c. in milk of broth emulsion of 1,000 million per 1 c.c. These were immediately fed on our vitamin B diet, namely, a suitably prepared enzyme-free wheat embryo preparation containing vitamin B. This was previously tested on pigeons and it was found that 1 grm. of this treated vitamin B food was sufficient to cure pigeons with head retraction, and certainly in the case of our rats that were almost dying from a vitamin B deficiency diet it was extraordinary to see how they revived on this food. Rats which were paralysed and too ill to drink milk, immediately this preparation was placed in their cages, either by instinct or by smell crawled along to it, and those which were too weak to dip their heads in it licked the jaws of those rats which were already eating the food. In a few hours they were quite lively. We were unable to culture any *Bacillus coli* from our original four weeks deficiency rats but we were able to culture *Bacillus coli* (the slides which I have under the microscope) from the nine weeks deficiency rats. Our next investigation was to investigate streptococcal cultures of deficiency and normal rats, and another ten normal and ten deficiency rats were fed with a streptococcus of the para-odontal type and the long form taken from the teeth of one of my patients. 1 c.c. of a 500 million broth culture was given in milk to each rat. On the fourth day the animals were killed by chloroform in the usual way and I now show the sections on the epidiascope.

It is interesting to note that none of these animals died from acute septicæmia or showed the least signs of being ill other than with the symptoms attributable to a deficiency diet. None of the rats had rigors and none of them developed any lung trouble. It was then decided to have some record of the peristalsis, and owing to the difficulty of fitting up shafting and drums in the laboratory it was decided to depend upon evidence derived from X-rays. These I think show quite conclusively that intestinal stasis is marked in the deficiency animal as compared with the normal. Barium sulphate mixed with white bread and made into pills with milk was fed to deficiency and normal rats. The first photograph was taken 3½ hours after a meal; second, 23 hours after a meal; third, 47 hours after a meal; fourth, on the fourth day.

You will note the enormous distension of the stomach in the deficiency animal as compared with the normal. You will also note as in all cases of dilated stomachs there is at first a rapid emptying of the bowel, presumably a reflex action; then follows a delay. This, I consider, is one of the most important findings of our investigations: that although we could not say that the animal is constipated, yet there was still present on the fourth day that which should have been passed on the third. Similarly, with patients, although they may not be constipated they tell you they have an action of the bowels every day. Yet, in all probability, they are passing to-day what ought to have passed yesterday.

As this is only a preliminary paper I will now summarize what I consider to be the interpretation of the findings of this extremely strenuous and tardy investigation.

A vitamin B deficiency in the diet of animals leads to a lowered vitality as shown by the subnormal temperature; secondly, a deficiency animal is prone to infection. On my farm where I have some 2,000 animals there is hardly one death per week, whereas in our animal house at University College, where I have 100 rats or so at a time, ten deaths might take place in a day. The rats die of acute toxæmia; the absence of peristalsis is due, in my opinion, to a paresis of a nerve supply followed by the atrophy of the muscle, with consequent distension and absorption of toxin. The track of infection is by way of the lymphatics and not by way of the blood-stream.

I was somewhat led astray in my researches owing to McCarrison showing us at his lectures in this building some time ago organisms in the blood-vessels in his deficiency monkeys; but, of course, one does not know how long after the death of the monkey these specimens were removed from the body, and we all know how rapidly organs become infected after death. We have been extremely careful in all our investigations to remove all specimens from our animals within a few minutes of cessation of life. In the sections from the animals

46 Rowlands: *Rheumatoid Arthritis: Is it a Deficiency Disease?*

you will see, seemingly, streptococci in the villi or glands, whereas the blood taken from the heart is sterile. It takes some time for organisms to follow this tract, but I believe that the disease of rheumatoid arthritis is resultant upon the toxæmia rather than upon a direct *Bacillus coli* infection. One of the marked symptoms that I have noticed amongst rheumatoid arthritis patients is the excessive fat in the majority of these cases, due I think, to thyroid deficiency, for when they are given thyroid they rapidly lose weight. It is possible that there is a hormone deficiency resultant upon the toxæmia. I am inclined to think that infection does not take place through the mouth in cases of bad teeth, but that organisms are swallowed and find their way into the intestine and obtain entrance to the system in this way. There is another important symptom that I have noticed amongst the diabetic sent to me for examination—namely, whenever I have been able to secure the passage of urine into a sterile vessel in every case *Bacillus coli* has been present. Also, in a number of cases of high blood-pressure, recent investigations have demonstrated the presence of *Bacillus coli* in the patients' urine. Is it possible that the toxæmia following upon deficiency is responsible for the onset in these cases? Within the last few years I have watched four cases with blood-pressure ranging from 200 to 250 mm. The patients were immediately placed upon a vitamin B excess diet and given injections of a vaccine prepared from their own urinary *Bacillus coli*. In every one of these the blood-pressure now remains in the region of 150 mm. and is constant. There is no doubt in my mind that the diet of the majority of human beings is wholly deficient in its vitamin B content. There is no complete absence of vitamin B; if that were so I am certain the population would rapidly diminish. Some years ago I investigated a large number of skeletons which were stated to be Saracen remains, and of some 100 bones that I was allowed to examine not a single one showed changes similar to those we find in rheumatoid arthritis to-day. In my opinion, rheumatoid arthritis, like many other diseases, is a disease of civilization. In days gone by the diet was entirely different, there was no such thing as white bread, no such a thing as feeding our milk cows on margarine by-products, our bullocks were not fattened on foods which are deficient in vitamin B. Our eggs were not produced by hens which are fed on deficiency foods, in fact there is very little in our diet to-day that is rich in vitamin B. In all probability we are fairly safe with a fairly sufficient amount of fat-soluble vitamin.

Taking the rat's life as lasting three years and that of the human subject at an average of sixty years, I must multiply my findings by twenty, and if it takes a rat some thirty weeks to develop signs of deficiency a human being will be between 20 to 25 years of age before any symptoms might occur. But according to my findings it is somewhere in the region of sixty weeks that I observe the most marked change; this when multiplied by twenty brings us to that period of life in the human being at which rheumatoid arthritis is most common. I am well aware that we frequently come across a very serious form of rheumatoid arthritis, the adolescent form, at a much younger age, but the most common type is that occurring between the ages of 40 to 50, and then the next most common, which I call the senile type with marked deformity and little pain, coming on about the age of 60. The point which I feel sure is uppermost in our minds is, what is a proper amount of vitamin B of some specific known material that is sufficient to prevent the changes which I have described in my experimental animals? It was difficult at first to find a material which was palatable and fairly constant in its vitamin B content. Whole-ground wheatmeal was first of all tried; the loss of a number of animals proved this to be very toxic. Next, yeast was tried and it was found to be unpalatable; was not always constant in its vitamin B content, and thus could not be standardized. Wheat germ was then tried; this was found to be extremely toxic and I therefore consulted Professor Mellanby of Sheffield, who stated that his results were similar to mine. But after various experiments the toxicity of the embryo was overcome and my standard vitamin B content is now a detoxicated wheat embryo which keeps almost indefinitely and is constant in its vitamin content. I have myself been taking this now for about two years without ill effect, in fact the results have been more than I ever anticipated. One tablespoonful daily is sufficient for an adult, and for a child two teaspoonfuls per day will yield the desired result. Milk of course, at one time, when our cows were properly fed, was a great source of vitamin B for our children, but to-day it is only for a short period that our cows are fed on any food which is likely to benefit the milk derived for this vitamin B. I am hoping that Professor Plimmer will give us some figures which will prove the fallacy of people who think that if they eat one or two pieces of wholemeal bread they are saved from all

diseases. We know that 2 per cent of detoxicated embryo is not sufficient even to keep the body together—at least 4 per cent. in my opinion is required, but these figures I will leave to Professor Plimmer. The conclusions at which I must arrive is that a large number of present-day ailments can be explained by the sections and specimens that I shall be exhibiting for your opinions this evening. Lastly, vitamin B is not cumulative, as in the case of the fat sol.

Relationship Between Rheumatoid Arthritis and Vitamin B Deficiency.

Now, what is, if any, the relationship between rheumatoid arthritis and a vitamin B deficiency? I have taken you a long way round to come home, but had we taken a short cut home there would have been very little left for discussion. Most of our cases of rheumatoid arthritis occur amongst our female patients; in about 80 per cent. of the cases sent to me the patients are women. We know that for various reasons intestinal disorders are more common in women. The period at which the disease sets in is fairly constant, namely, between the years of 40 to 50. This is the age at which deficiency would show itself most.

Similarity of Vitamin B Deficiency to a Nervé Disease.

We who work with animals all know that one symptom constantly present in vitamin B deficiency is paralysis. In rheumatoid arthritis a joint commonly involved is the knee; before the pain becomes centralized in the knee the patient will, in most cases, complain of tingling or numbness around the joint; wasting of the vastus internus sets in, and there are marked areas of hyperæsthesia above the knee-joint, and also on the outer part of the leg. We know that the nerve to the vastus internus supplies the knee-joint, or at least, its synovial membrane. There is no wasting of the rectus.

The Shoulder-Joint.—The patient often complains of pain over the deltoid before there are any marked bony changes, and if you try the sensitive areas with a sharp pin you would always pick out that area of skin which is supplied by the cutaneous branch of the circumflex. The circumflex, of course, gives a large branch to the joint as a trophic nerve.

The Hand.—The marked wasting of the interosseous muscles and of the thenar eminence supplied by the deep portion of the ulnar-nerve. There is never any wasting of the hypothenar eminence. If the wasting were due to disuse why should some muscles be involved and not others?

Ætiological Conclusions.

(1) The absence of any organism in the blood, in the joints and in the tissues of the patient is strongly in favour of the disease being of toxic origin. The high percentage of cases, namely, 96 per cent., in which there is bacilluria, constitutes strong evidence of the disease being the results of bacterial products.

(2) Other conditions accompanying rheumatoid arthritis are the distended stomach, constipation, etc.

(3) The similarity in the anatomical change in almost all cases is remarkable. Some radiograms of the feet will illustrate this.

(4) Injury is a predisposing cause as to which joint will be involved.

(5) The disease is one of civilization and on the increase, such increase being consistent with the changes in our diet during the last 100 years. First, due to bread; secondly, due to the introduction of cake feeding to our stock.

Conclusions as to Treatment.

I see two members of our profession in my audience who consulted me for acute rheumatic pains. A sample of urine was taken from them and was found in both cases to be teeming with *Bacillus coli*; they were placed on a concentrated vitamin B diet and they soon got well. From one of the patients we were not able to secure a second specimen of urine; from the other a second specimen was procured and immediately the pain ceased, the urine was sterile on culture on every occasion.

We all know by experience that massage, electricity, thermal and drug treatment have done but little to help our patients. My results justify the form of treatment that I advise. First, look for all sources of infections, in the nose, throat, teeth, sinuses, urine, stools, etc., and remove these as far as possible; secondly, immunize our patients against the results of infection, and above all place them at once on an excess vitamin B diet.

Lastly, to quote the words of Professor Willstätter mentioned in his recent Faraday Lecture, "if what I have done, be it right or be it wrong, leads to a discussion that may lead to further research work, then I feel I have achieved something."

48 Rowlands: *Rheumatoid Arthritis: Is it a Deficiency Disease?*

Dr. ROWLANDS then showed a series of slides by means of the epidiascope:

(1) Normal cæcum of the rat, the section having been cut in the region of the ileo-cæcal valve (fig. 1).

(2) Cæcum of a thirty-five-week rat, × 10.

(3) Thirty-five-week rat on a partially deficient-vitamin diet (fig. 2).

(4) A rat after eighty weeks' deficiency diet (fig. 3). That rat was given 2 per cent. marmite. At this stage, staining for organisms was not thought of; the search was for intestinal stasis. This showed that the whole cæcum had changed and that the muscle had wasted.

(5) No. 1 animal with normal cæcum, × 120.

(6) The cæcum of a rat on thirty-five days of deficiency diet, × 120 (fig. 4). This showed marked changes, not only in the mucous membrane, but also in the areolar tissue of the two outer muscles, and in the circular muscles.

(7) The cæcum of a rat after eighty weeks' deficiency feeding, × 230, and next the normal cæcum of a rat × 230 (fig. 4A).

(8) The whole cæcum of a rat fed for thirty-five weeks on a partially deficient diet, × 230. The muscular tissue and mucous membrane of the normal animal were twice this dimension.

(9) A normal colon, × 15 (fig. 5). Not less than fifteen or twenty animals were killed at a time, and every third animal was examined in order to ensure accuracy.

(10) A colon after thirty-five weeks' deficiency feeding, × 15 (fig. 6). Already changes in it were beginning to be marked. Animals could not be kept alive on a diet which was completely deficient in vitamins.

(11) A colon after eighty weeks of partial deficiency feeding (fig. 7).

(12) The colon of a normal rat, exhibiting the mucous membrane in more detail, × 150.

(13) The colon of a rat fed on a partially deficient food, × 150. Already changes were taking place, even in the muscle.

(14) An eighty-week deficiency-fed rat, showing more marked changes taking place, × 150.

(15) The same magnified 500 times, distinctly showing the cell-disintegration.

(16) A series of skiagrams of deficiency-fed and non-deficiency-fed animals. The first was of a normal stomach, fed with barium sulphate, taken three and a half hours after the meal (fig. 8). Next, for comparison, a rat fed on a deficiency diet for twelve weeks only (fig. 8A). The stomach had dropped and was distended to about three times the normal. Rat had recovered after proper feeding, and was exhibited at the meeting. It had been on full vitamin B diet for the past fortnight, and the shininess of the coat was returning, the animal being well and energetic.

(17) A skiagram taken twenty-four hours after a meal showed an expulsion more rapid than that occurring normally (figs. 9 and 10). Dr. Finzi had said that in the case of these dilated stomachs there was first a reflex irritation and a rapid expulsion of contents, and then delay.

(18) A normal rat at forty-eight hours; no food was seen in the rectum, nor in the descending colon (fig. 11).

(19) An abnormal rat at the same interval; it passed the bismuth on the fourth day, whereas in the normal rat none remained after the third day (figs. 12 and 13).

(20) With regard to the question of micro-organisms, he next showed an animal which had received an admixture of pneumococci in its food. The lymphoid tissue was full of micro-organisms.

(21) The intestine of a rat fed for twelve weeks on a deficiency diet and micro-organisms. He showed a view (× 1,000) in order to demonstrate the micro-organisms. Not only had the micro-organisms found their way into the tissues, but they were growing there. Mere feeding would not have given such good preservation of the organisms. The streptococcus was much easier to demonstrate than the pneumococcus or the staphylococcus (figs. 14 to 17).

(22) A series of about one hundred skiagrams of the feet of cases of rheumatoid arthritis were then shown, and in every instance the osteophyte indicated some part which bore the stress of walking, mostly the inner side of the great toe. He believed it was a question of nerve irritation.

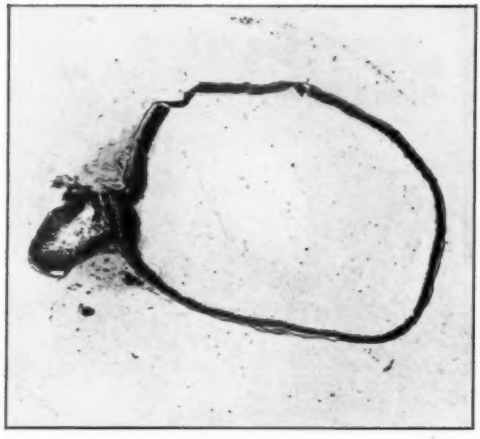

FIG. 1.—Normal cæcum.

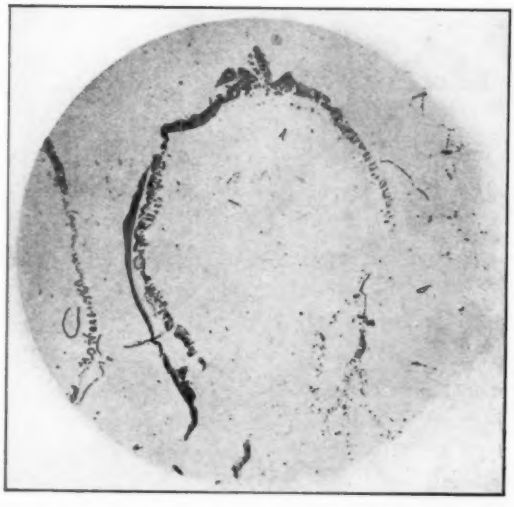

FIG. 2.—Cæcum of thirty-five weeks' partial deficiency.

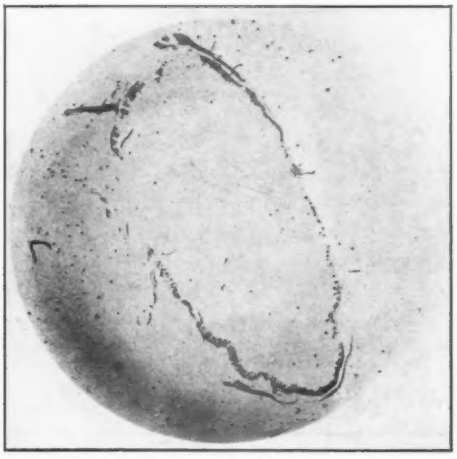

FIG. 3.—Cæcum of eighty weeks' partial deficiency.

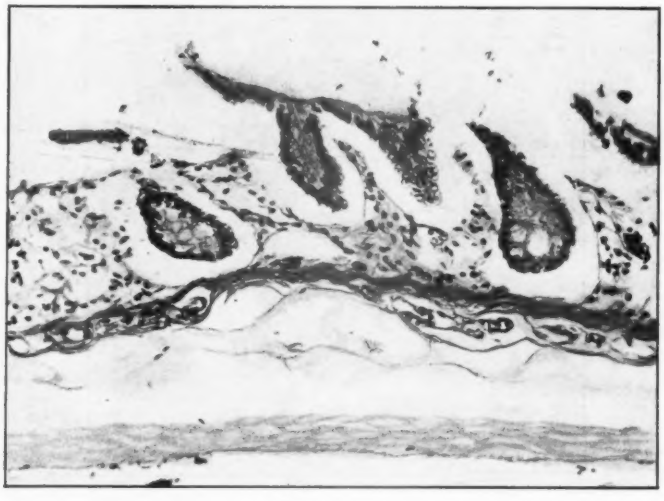

FIG. 4.—Section of cæcum of thirty-five weeks' partial deficiency rat, × 230.

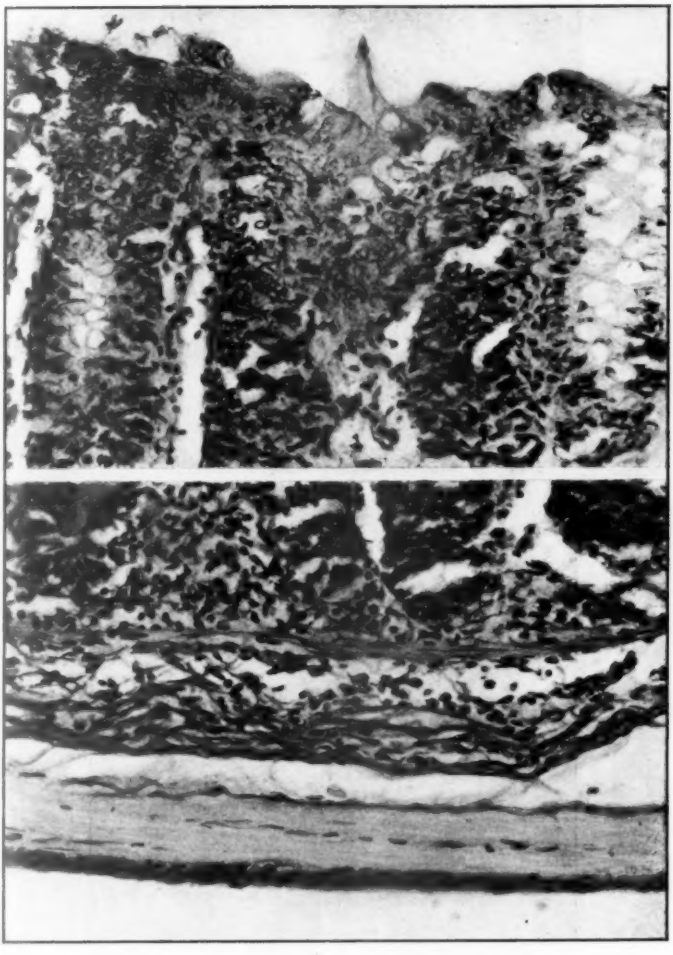

FIG. 4A.—Section of cæcum of normal rat, × 230.

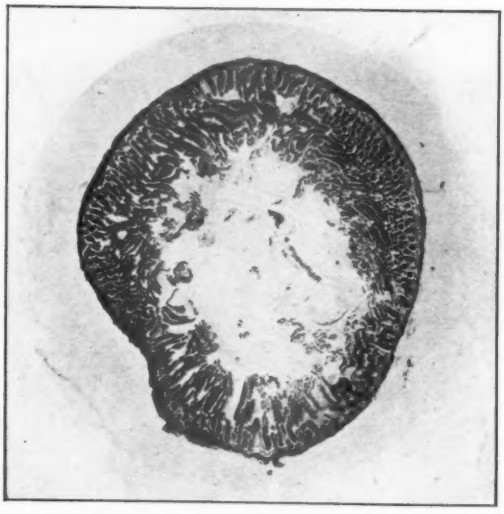

FIG. 5.—Section of normal colon, × 15.

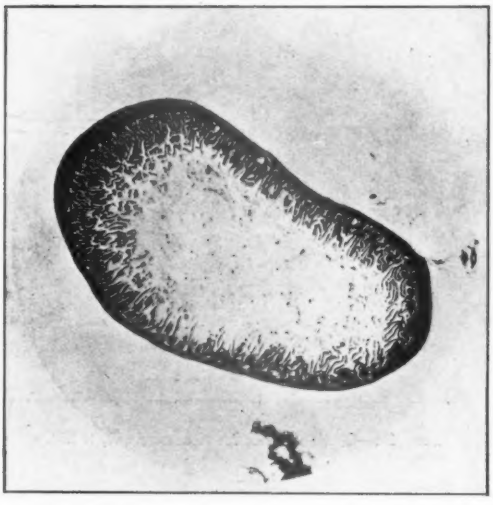

FIG. 6.—Section of colon in thirty-five weeks' partial deficiency rat, × 15.

FIG. 7.—Section of colon of eighty weeks' partial deficiency rat, × 15.

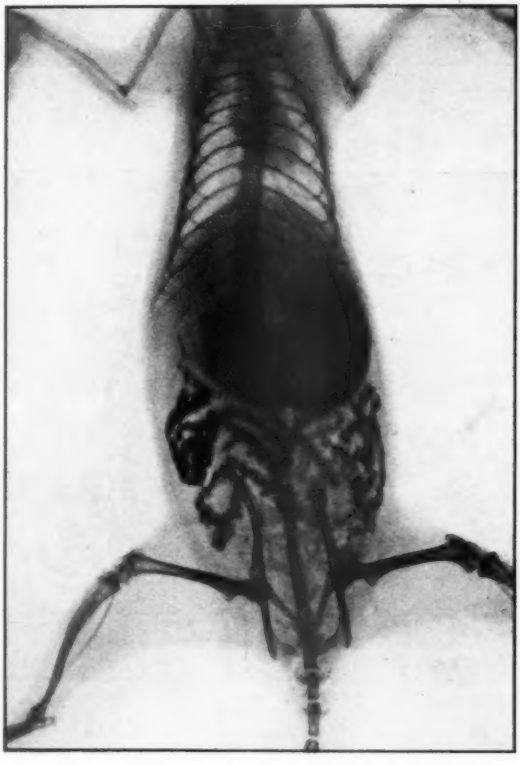

FIG. 8.—Stomach of normal rat three and a half hours after barium meal.

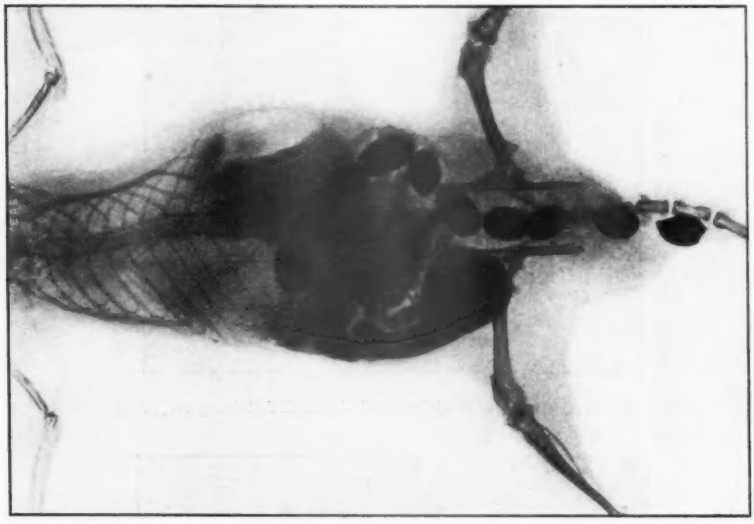

FIG. 9.—Deficiency rat twenty-four hours after meal.

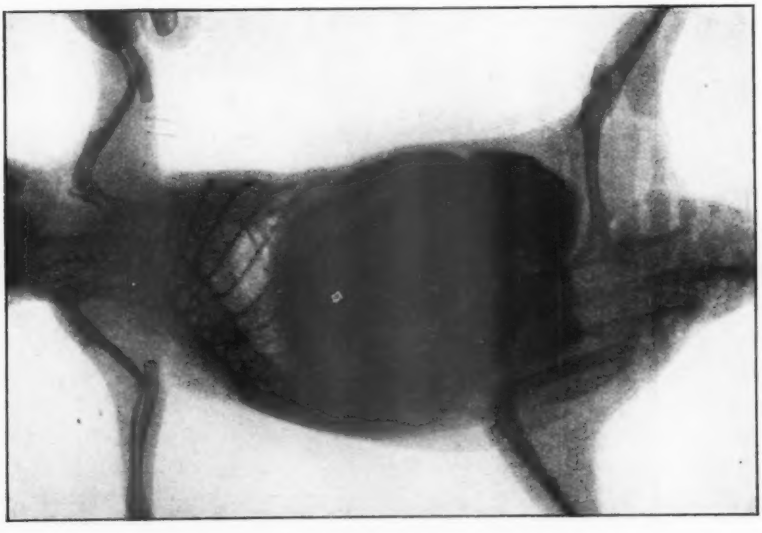

FIG. 8A.—Stomach of rat three and a half hours after barium meal in twelve weeks' partial deficiency rat.

Section of Comparative Medicine 55

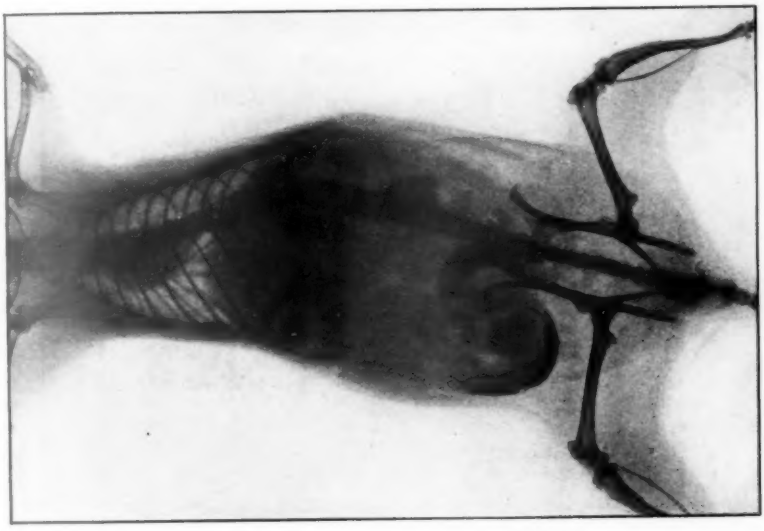

FIG. 11.—Normal rat forty-eight hours after meal.

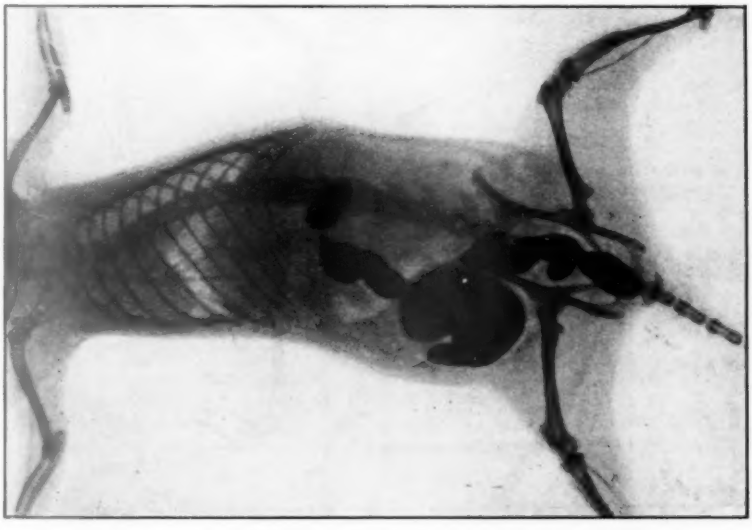

FIG. 10.—Normal rat second day, twenty-four hours.

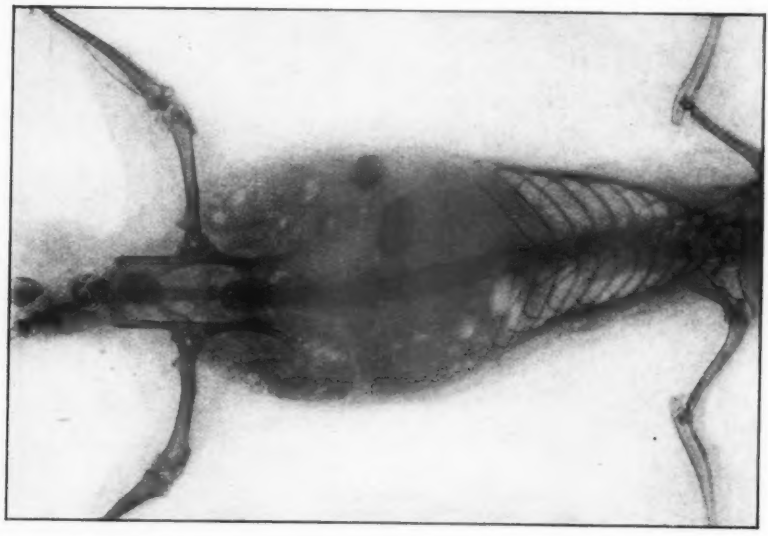

FIG. 13.—Abnormal rat seventy-six hours after barium meal.

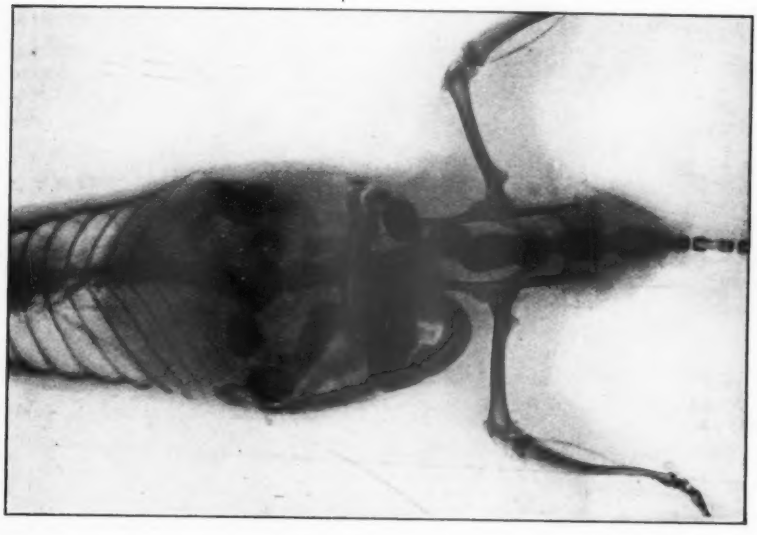

FIG. 12.—Abnormal rat, forty-eight hours after meal.

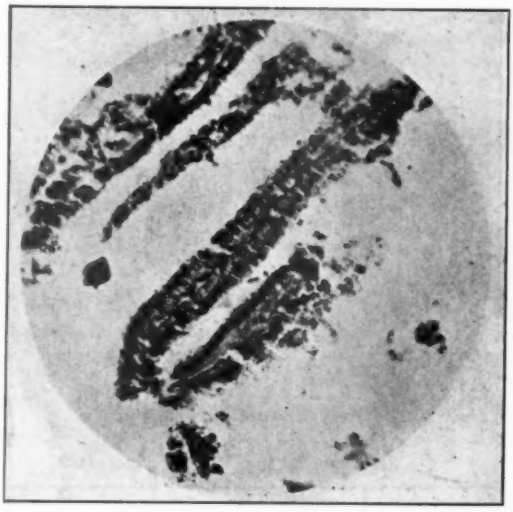

FIG. 14.—Section of intestine, deficiency animal, showing streptococci in lacteal, × 200.

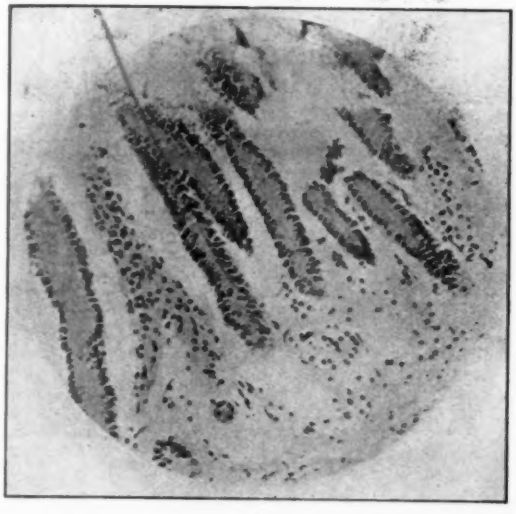

FIG. 15.—Section of intestine of normal animal, having been fed on streptococci.

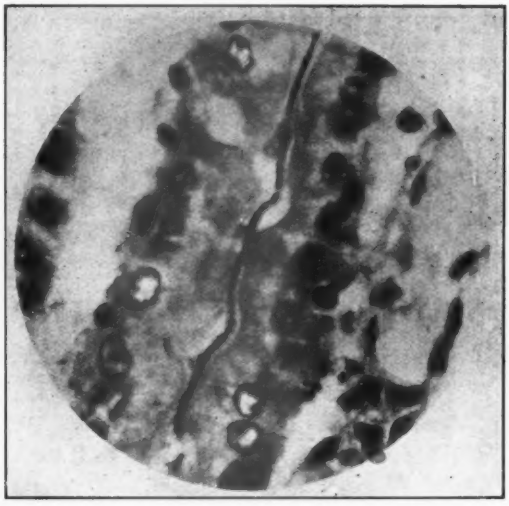

FIG. 16.—Twelve weeks' deficiency-fed rat showing streptococci, × 1,000.

FIG. 17.—Twelve weeks' deficiency-fed rat showing streptococci, × 1,000.

Section of Comparative Medicine 59

[*June* 2, 1927.

Discussion.—Dr. ETHEL BROWNING said that the histological and bacteriological findings in the course of this work on rats had been very definite and conclusive. Professor Swale Vincent had kindly examined some of the slides which Dr. Rowlands and herself had considered the most important and had confirmed their opinions in each case.

In the beginning they had intended to concentrate chiefly upon the cæcum, as being that part of the intestine in which changes due to B-vitamin deficiency would probably be most in evidence, but they had soon found that the rest of the intestine shared in these changes, and therefore they continued their investigation on the cæcum and large and small intestine.

There was one small point in the actual dissection of the rats which Dr. Rowlands had asked her to mention. In making cultures of the urine of normal rats they had found that, considering the small size of the animals, the best way to secure any urine for culture was to remove the whole bladder by cutting just below the neck with sterile scissors and dropping the whole organ into a tube of broth, which was incubated for twenty-four hours and then plated on to MacConkey's agar. In the ordinary way one would naturally expose the bladder by opening the abdomen in the middle line, but they suspected that by this method the outer surface of the bladder was likely to come into contact with falling hairs from a region sure to be highly infected with the organisms consistently found in the cultures from these particular animals. She had therefore opened the rat by a rectangular incision commencing at the right side, immediately below the thorax, cutting straight across and down both sides, holding the apron-flap so produced well away from contact with the abdominal contents, and pinning it down so that no external infection could possibly reach the bladder. Using this method they had found that in every case, in the rats fed on normal diet, the urine was sterile.

After the bladder had been removed aseptically, the presence of peristalsis was looked for. In normal animals it was strongly marked, the waves passing regularly without any stimulation, but in the deficiency-fed rats it was practically absent, only a few feeble waves being elicited by tapping the intestine. From every animal a portion of the transverse colon, and one of the cæcum, from just below the ileo-cæcal valve to within a quarter of an inch of the blind end, were removed and placed in 50 per cent. alcohol, then (through successive stages) in absolute alcohol, equal parts of chloroform and carbon bisulphide, and lastly in paraffin wax, melting point 63° F. Then, after blocking, sections were cut with a Cambridge rocking microtome and stained according to the examination required.

For ordinary tissue staining hæmatoxylin and eosin were used, for comparing the thickness of the muscular coats hæmatoxylin and van Gieson, and for showing the presence of organisms in the tissues Gram's iodine, counter-staining with neutral red. In all the deficiency-fed animals the same difficulty in cutting and staining had been experienced; all the sections were more fragile than in the normal animals, and all showed some deficiency in staining.

In the cæcum and colon of the animals fed on an emulsion of organisms the greatest difficulty encountered was that of preventing over-decolorization of the organisms. It was found necessary to avoid using alcohol, and to decolorize with a mixture of aniline oil and xylol. In making use of the intestines of pigeons and chickens, kindly sent to them by Professor Plimmer, it had been impossible to try to demonstrate the presence of organisms, since a fairly long interval had usually elapsed between the death of the animal and their reception of it. On this account, too, the tissues were difficult to block and cut successfully, but in all cases of deficiency-fed animals the same general histological changes in the intestinal wall were found to be present.

One or two other points might be worth emphasizing in relation to the effects—macroscopical and microscopical—produced by B-deficiency feeding:—

(1) Rats fed on absolute B-deficiency for three weeks were small and in poor condition, but had not developed symptoms of beri-beri. Intestines (macroscopically) thin, flabby and shrunken, cæcum not dilated. No trace of abdominal fat present. Microscopically, chief changes shown in mucous and areolar tissue—loosening and vacuolation. No organisms found.

(2) Rats fed for a long time on a B-deficiency diet, containing a sufficient percentage of marmite to keep them alive, developed an abnormal amount of abdominal fat. (This was seen also in hens fed on marmite, 5 per cent.) Peristalsis was again poor or absent, and microscopically, the cæcum, more strikingly than the colon, showed the same changes in structure as were found in absolute deficiency rats. In these long-standing cases micro-organisms (chiefly diplococci) were found in the mucous and areolar tissue.

60 Rowlands: *Rheumatoid Arthritis: Is it a Deficiency Disease?*

(3) Rats fed on a very small percentage of marmite, followed by a period of absolute deficiency, showed very rapid deterioration, accompanied by paralysis and lack of orientation, almost complete loss of appetite, diseases of skin and eyes, and complete absence of growth. On their being supplied with vitamin B, recovery was extraordinarily rapid, with return of appetite, increase of weight, and remarkable improvement in general condition.

Professor R. H. A. PLIMMER said he could not, as a simple chemist, discuss Dr. Rowlands' interesting paper from the medical and pathological side. Dr. Rowlands' results were wonderful, and must open the way to many other aspects of chronic disease in connexion with vitamin deficiency.

He had known of Dr. Rowlands' work for a long time and had felt it gave just that final touch to the observations of Dr. Rosedale, Mr. Raymond and himself, made during the past six years, on the balance of food by vitamin B. *Their experiments had been made upon birds and rats, and the possibility of applying the conclusions to human beings had been questionable; the research and clinical experience of Dr. Rowlands supplied that missing link which justified the belief that the effects of a diet containing some*, but *not enough*, vitamin B, were the same in birds, rats and man.

Last year he (the speaker) had the pleasure of bringing their results before the Society. They had used a diet in which dried yeast was the only source of vitamin B, and had found that the minimum quantity of dried yeast for the maintenance of birds was 4 per cent. of the *dry* weight of the food.

On 1 per cent. of dried yeast the birds died quickly from polyneuritis, i.e., beri-beri. On 2 per cent. they lived a long time but were obviously very unhealthy. On post-mortem examination they were found to be loaded with fat, and to the naked eye the intestines and appendices were abnormal. Their hearts were enlarged. Dr. Pulvertaft had some specimens of these hearts to show and would describe the pathology.

On more yeast, 4 per cent., the birds lived for a longer time, but were not altogether normal. On still more yeast, 6 per cent.; they were quite fit.

It had been their object also to find out *how much* of ordinary foodstuffs was needed to maintain a bird (pigeon) for a long period, and the following data had been determined:—

Dried yeast, at least	4	per cent. (dry weight) to balance a diet lacking in vitamin B.
Wheat germ	6	" " " " "
Marmite	10	" " " " "
Peanuts	20	" " " " "
Peas, beans, lentils	30	" " " " "
Wheat, barley, rye	40	" " " " "
Almonds, chestnuts	40	" " " " "
Egg yolk	50	" " " " "
Oatmeal	90	" " " " "
Potato	90	" " " " "

Fruits, such as apricot, apple, or banana, would not maintain a bird, neither would vegetables, such as cabbage, carrot, or onion. These were all very poor in vitamin B. Their findings were not in accord with the high value assigned to these foodstuffs in the report of the Medical Research Committee on Accessory Food Factors. Neither could they agree with the statement made in this report that "The deficiency in white wheaten bread is under ordinary conditions made good by the varied diet enjoyed by Europeans."

Variety was no safeguard if the various foodstuffs were all poor in vitamin B. He (the speaker) had estimated that the ordinary everyday diet only supplied approximately two-thirds of our full requirement of vitamin B. We did not get beri-beri, but intestinal trouble and heart trouble were common troubles in almost every household.

To get the full supply of vitamin B in our diet, the only practicable way was to use wholemeal flour, not only in bread but also in cakes and puddings. The only alternative to wholemeal would be to add large quantities of marmite or wheat germ (temax) to the ordinary diet. One roll of wholemeal bread a day was not enough, it was not a magic charm but a question of quantity and balance. For this reason peas and beans might be excluded from the reckoning, as they were only occasionally eaten and then in small quantities. Again, one egg supplied little vitamin B. To derive an adequate supply from eggs half the diet would have to be eggs.

Meat and fish were very poor and required balancing. Potatoes and oatmeal only contained enough vitamin B to balance their own carbohydrate. Vegetables and fruit contributed a negligible quantity.

The requirements of birds and man for vitamin B corresponded closely. It was known that for birds 40 per cent. barley was required and for man at least 30 per cent. (From experiences with feeding the Japanese Navy.)

The effect of a continual slight shortage of vitamin B was stagnation of the contents of the intestine. It was the custom to remove the stoppage by pills or salts. During stoppage some sort of ptomaine was formed (from decomposing protein), and the daily absorption of a little of this toxic ptomaine produced the heart trouble and other troubles.

Professor J. C. DRUMMOND said that Dr. Rowlands' paper focused attention on that borderland of disease which those who worked on nutrition realized to be of such importance. It was an erroneous assumption, widely held and dangerous, that a moderately varied diet afforded protection from vitamin deficiency. It was easy to compile a varied diet sheet which might be very deficient in necessary vitamins. That fact had been well established by McCarrison's experiments on monkeys, and by Dr. Rowlands' researches. Those who worked on the purely scientific side of vitamin deficiency frequently imposed almost complete deficiencies, which inevitably produced a serious condition; and seldom used animals which had been partially deprived of vitamins. The seriousness of the condition produced in animals by a partial deficiency was illustrated by experiments on the fecundity of the rat; on such deficiency there were changes in the fertility and in the sex-ratio of litters born during a chronic condition of under-nutrition in the parents.

Thinning of the intestinal wall was an important fact to have established, and the easier penetration of that wall by micro-organisms in such under-nourished animals was very convincing.

Retardation of peristalsis in Dr. Rowlands' rats seemed to have been established, though this finding was not quite in accord with the results of his (Professor Drummond's) own experiments on pigeons. He thought that the explanation of the difference was that in the pigeon there was a rather complex nervous mechanism, by which the opening and shutting of the crop influenced the movements of the lower gut.

The position at present was somewhat complicated by the fact that the question of vitamin B was in the melting pot owing to the discovery of at least two factors being concerned in the physiological action of yeast. In the light of this discovery some of the accepted figures, such as those given by Professor Plimmer, might have to be revised. The antineuritic substance, the absence of which probably produced the changes Dr. Rowlands described, was very much more concentrated in wheat embryo than in yeast.

With regard to the relation of Dr. Rowlands' experiments to the question of rheumatoid arthritis, he asked whether the joints of the rats in question had shown arthritic changes. He asked this because, having observed the rats which Dr. Rowlands kept in the animal house at University College, it now occurred to him (Professor Drummond) that after being a long time on this deficiency they showed stiffness at the joints. He remembered having observed stiffness in the limbs of experimental rats fed on food deficient in vitamin A, yet for years no one thought of examining their joints. Had they done so the modern views on rickets would have been elaborated years earlier.

He thought the most striking result of these experiments was the clear proof that under this prolonged nutrition on a deficiency diet the animal was placed on the border-line of disease for a long time, so that its generally lowered resistance made it an easy prey to any infection to which it might be exposed.

Professor SWALE VINCENT said that the changes in the intestine following the prolonged shortage diet were very striking, and he could not understand how anyone who had examined the alimentary canal of animals which had been kept on such deficiency diet could have overlooked such definite atrophy. The effect, indeed, was precisely as if one had cut the nerves. Dr. Rowlands spoke of the "trophic nerves." Every nerve was a trophic nerve, and it would be difficult to know which change had come first in this case. Disuse of a muscle would cause it to atrophy; atrophy of a skeletal muscle was brought about more quickly by cutting its tendon than by cutting its nerve. Therefore in these cases the atrophy might have been caused by the stasis, and not the stasis by the atrophy. The changes were very evident, and it did not need an expert histologist to verify Dr. Rowlands' conclusions.

62 Rowlands: *Rheumatoid Arthritis: Is it a Deficiency Disease?*

Dr. Rowlands had referred to the passage of organisms into the lymph channels, and he had shown the speaker a slide in which, he thought, organisms were to be seen in the lacteal radicles of the villi. He (the speaker) agreed that this was where the organisms were situated, but unless one devoted a good deal of time to the examination of serial sections it was difficult to be sure about this. He thought the organisms showed signs of wandering along the lymphatics, rather than in the blood-vessels.

Dr. R. J. V. PULVERTAFT said that he merely had to report on certain changes in the hearts of some chickens experimented upon, for which purpose the hearts had been sent to him. He had received thirty such hearts. Four of them were from normal chickens, the other twenty-six were from chickens which had been fed for varying periods on deficiency diets. The earliest changes were seen after two months of deficiency dieting, the latest after seven months of it. In all the cases the changes were the same. In nineteen of the hearts no changes were shown, in seven the changes he was about to describe were in evidence. First, he looked for signs of fatty degeneration, as Professor Plimmer had noted that these hearts were very fat indeed. In sections cut and frozen and stained with Sudan 3, he never found fatty degeneration proper, but there was a marked increase in the amount of fatty tissue between the muscle fibres.

The significant pathological changes he found were seven in number in the animals examined. Three were cases of fibrous reaction, interstitial fibrosis and degeneration of heart muscle fibres. Five showed a condition to which he had given particular attention, and upon which he would like opinions to be expressed. He saw areas, roughly oval in shape, in which there still remained certain vestiges of heart muscle, atrophied and fibrosed.

These lesions were in relation to the blood-vessels. The cells of those were, for the most part, small lymphocytes, and with them were some large endothelial cells. It was simply an exudate between the heart-muscle fibres. In four or five cases there was only one lesion, in the fifth case there were many such lesions. In all but one case the lesions were in the muscles; in that remaining case they were present in the pericardium.

One or two points required discussion. As these animals were chickens, the question had arisen as to whether it was the infective leukæmia of chickens, but this disease could be excluded. The blood-vessels, in most cases, were crammed with cells, but there were only one or two white cells among the red, whereas the ordinary ratio in infective leukæmia of chickens was that of two red cells to one white cell; also in these particular chickens the liver condition seen in that disease was absent.

In rheumatic states the Aschoff body was described. He showed an illustration which exhibited the condition to which these hearts bore the closest analogy. It was that of a specimen taken from the heart-muscle in a case of Graves' disease, which was illustrated in *Brain*, in 1925. And there was a close relationship with myasthenia gravis.

The next slide showed a section from an eye muscle. It was said that these lesions were difficult to find, and that in many sections they were missed; he had himself missed them in two or three of the series, but had picked them up on entering further into the section. In preparing sections in the future it would be necessary to examine, especially, the thyroid gland, and muscle other than that of the heart, for the presence of lesions, as these other lesions might prove to be very important.

Professor R. A. PETERS said his experience in this connexion was entirely with pigeons as a test for the concentration of the anti-neuritic factor in vitamin B, i.e., one of the factors to which Professor Drummond had alluded. There were only two points which he could usefully make in this discussion.

He supposed it was fairly clear that the general population suffered in a greater or less degree from vitamin deficiency, and that being so, the question arose as to why a certain proportion suffered from rheumatoid arthritis, while the others did not. It argued some essential difference in the two classes. In the work which he and his colleagues had recently been doing, it had happened, on one or two occasions, in working on yeast concentration, that on arriving at a time when there was a purer substance, the activity was greater than in the original yeast. There seemed to be something which was cleared out, something which exerted an inhibition on the vitamin. That might possibly be connected or correlated with the availability, in the intestine, of the material. It was a question whether digestion could play a part in selecting these persons in a special way if their condition

was due to vitamin deficiency. Was it possible that certain people could not normally digest certain of their food materials, so that they had not the benefit of a due absorption of those factors?

The other point which occurred to him was concerned with bacteria. He thought it was an established fact that particular bacteria would synthesize vitamin B. Other bacteria probably would destroy it. The question whether, even if vitamin was present in the intestine, it was properly used, in the presence of the intestinal bacteria, must be an important one.

Dr. VINCENT COATES (Bath) said that Dr. Rowlands had not made himself clear as to which types of non-suppurating arthritis he was referring to. "Rheumatoid" was a generic term. He had not understood whether Dr. Rowlands denied the question of infection. If this were so it would mean denial of the usual significance assigned to pyrexia, hot swollen joints, enlarged glands and spleen, and leucocytosis.

Dr. Rowlands had made a valuable suggestion that vitamin deficiency might be one of the missing links in the preparation of the soil for infection, and he (Dr. Coates) would, in future, consider each case most carefully with regard to the vitamin deficiency factor.

He asked what was found in the joints of the rats which had been experimented upon. If those rats had joint conditions similar to those found in non-suppurative arthritis, it was very good evidence that there was an association between the deficiency factor and the arthritis, but if such changes were absent he failed to see the connexion between rheumatoid arthritis and vitamin deficiency.

As to whether or not infective arthritis was a blood infection, the evidence was all in favour of its being so, because the histological characters of a subcutaneous node from such cases showed that the node was due to a vascular lesion.

Dr. W. CRAMER said that one of the most characteristic changes which occurred when there was a deficiency of vitamin B was the change in the lymphoid tissue. He suggested that Dr. Rowlands should have blood-counts made both in his animals and in his patients, in order to discover whether there was a lymphopænia; he (the speaker) and his colleagues had observed it regularly in incomplete vitamin-B feeding. A decrease in temperature was also a characteristic change. Possibly the infection which Dr. Rowlands had observed was a result of this fall in temperature; animals which had been previously resistant to infection could be infected if means were adopted to lower their temperature. In ordinary vitamin-B deficiency, infection did not occur; in this respect it was different from vitamin-A deficiency, for in that condition there was always infection.

The statement that vitamin-A deficiency diminished resistance to infection was only partly true. Dr. Kingsbury and himself had carried out experiments in which bacteria had been subcutaneously injected into animals with vitamin-A deficiency. These animals had been able to dispose of the bacteria as well as normal animals did; both were tested with regard to the production of bacteriolysins and there was no difference. Nevertheless, all these animals became infected spontaneously without any feeding with bacteria. He thought that the mechanism responsible for this infection was a defective mucous membrane activity. With vitamin-A deficiency, but not with acute vitamin-B deficiency, there was a very definite atrophy of the secretion of mucus from the lining membranes of the respiratory tract and from the alimentary tract. Two or three years ago he had shown, at a meeting of the Society, slides of the intestines of rats kept on a vitamin-A deficiency diet in which the bacteria could be seen crawling into the empty mucous cells of the intestine. One of the most characteristic and constant lesions in vitamin-A deficiency in animals was an infection of the lungs, and there was also disease associated with atrophy of the lachrymal glands of the eye. Some of the preparations now shown by Dr. Rowlands so closely resembled those which he (the speaker) had shown as resulting from a diet deficient in vitamin A that there might have been in Dr. Rowlands' animals a combined deficiency of vitamins A and B. He (Dr. Cramer) wondered whether Dr. Rowlands had given cod-liver oil abundantly to these animals or whether he had relied entirely on butter-fat.

On the whole, Dr. Rowlands' observations were in striking agreement with those of Colonel McCarrison and himself, some of which had been placed before the Society.

Miss H. CHICK said that after listening to the discussion, she felt, as one who was engaged in research upon the relation of vitamin B to nutrition, that there was need to emphasize the fact that our knowledge of the subject was still very uncertain and incomplete. One should be very cautious in applying the results obtained from the study either of

64 Rowlands: *Rheumatoid Arthritis: Is it a Deficiency Disease?*

polyneuritis in birds or of growth in rats to the problems of human diets, and at the present time it was premature to urge any radical changes in human dietary on the basis of such experiments. It was now established that what had previously been called vitamin B consisted of certainly two, perhaps three, different things, and that the substance which cured the polyneuritis of pigeons was only part of what people working with rats had called vitamin B, or water-soluble B.

Evidence had come from the work of Goldberger and his colleagues, in the United States, that one constituent of the so-called vitamin B, other than the antineuritic vitamin, was concerned in the prevention of human pellagra. It might also have much to do with the maintenance of the health of the alimentary canal. Attempts to separate the constituents of vitamin B had led her to believe that the pathological condition of the alimentary canal seen in animals which had been deprived of vitamin B was, probably, not due to the absence of the constituent which prevented the occurrence of polyneuritis in pigeons. This other constituent had not yet received a special name.

It was important at the present time to discourage dogmatic statements which, emanating from learned societies, might get into the lay papers and possibly mislead the public. The right course was to await the results of the various researches on the subject which were now being conducted on both sides of the Atlantic.

Dr. TOM HARE said that the value of Dr. Rowlands' animal experiments *per se* had been estimated by those qualified to express opinions upon the vitamins; it was upon their correlation with certain clinical observations of human arthritis that he (the speaker) would offer a few remarks.

He understood Dr. Rowlands' main proposition to be that rheumatoid arthritis was due to absorption of toxins from a large intestine, the resistance of which had been destroyed by a vitamin-B deficient dietary. The data upon which this notion had been founded were (1) experimental, and (2) clinical. Thus, in the first place, Dr. Rowlands found that experimental animals reduced to a state of ill-health by vitamin-B deficient dietary, revealed evidence of certain retrogressive changes of the large intestine, together with a bacilluria; and secondly, that a bacilluria was found in 100 per cent. of a series of human arthritics. He gathered that these two series of observations were further correlated upon the successful removal of the bacilluria, both in the animals and the arthritics, by vitamin-B therapy.

He asked whether in the case of the human patients, controls by means of other arthritic therapeutic agents for the removal of the bacilluria, e.g., foreign proteins, hormones, heat, massage, etc., were tried? Might he conclude that Dr. Rowlands did not regard his course of anti-bacilluric therapy as specific? Did he suggest that a bacilluria was invariably present in rheumatoid arthritic patients? If so, when, in the course of the arthritic disease, did the urinary changes begin? If the notion were correct that the arthritic process was due to the secondary intestinal toxæmia, then the onset of rheumatoid arthritis must invariably be preceded by a period of ill-health; but was this in accord with the experience of clinicians?

These questions required a definition of rheumatoid arthritis. Dr. Rowlands had given some evidence of the ages of his patients, and of the X-ray pictures of some of the affected joints, and upon that evidence he (the speaker) would suggest these patients were manifesting the connective-tissue metaplasia characteristic of osteo-arthritis. In a recent publication[1] the strength of Sir Archibald Garrod's notion of osteo-arthritis as the end-result of various forms of active arthritis had been emphasized; and an endeavour had been made to show that the hyperplastic, metaplastic, and atrophic changes known as osteo-arthritis, were the sequelæ of any of the agents (e.g., bacteria, toxins, trauma, etc.) known to be capable of effecting local injury in the vascular connective tissues. To this list of causes of osteo-arthritis the rheumatoid factor was added, the term "rheumatoid arthritis" thus being reserved for those cases which manifested the accepted clinical phenomena of rheumatoid arthritis, but for which no specific cause had been determined. It was only rendering a difficult problem more confusing to include as rheumatoid arthritis cases of acute or subacute arthritis and fibrositis, the causal agent of which might be shown to be a known bacterium, toxin, etc. For instance, the signs typical of rheumatoid arthritis might arise because a streptococcus had escaped into the connective tissues from a septic throat, and yet pass off if the primary focus

[1] Hare, T., "An Investigation of the Etiology and Pathogeny of Equine Chronic Arthritis (Rheumatoid Arthritis)," *Veterinary Record*, 1927, vii (n.s.), 411 and 431.

was quickly cleared up—such a case belonged to the infective group of the known specific arthritides.

So long as clinicians were able to differentiate a distinct type of arthritis from the arthritides of known specific cause, and so long as its causative factor remained undetermined, its retention under the term "rheumatoid arthritis" was justified; consequently he (the speaker) defined rheumatoid arthritis as a disease of the vascular connective tissues, tending to chronicity, and probably specific, but due to no specific cause yet known.

He (the speaker) had also endeavoured to show that the pathogenic activities of the rheumatoid factor in the vascular connective tissues were of sudden onset and of transitory duration; and might manifest themselves (i.e., the so-called clinical exacerbations) in the old foci or in new foci by the cardinal signs of inflammation. Therefore he would suggest first, that it was only at the onset of the initial attack—or of an exacerbation—that investigation into the ætiology of rheumatoid arthritis could afford reliable information; and, secondly, that when Dr. Rowlands' patients presented themselves for examination, the causative factor, assuming it to be the rheumatoid, had long disappeared.

Assuming his (the speaker's) definition to be correct, and this osteo-arthritis manifested by Dr. Rowlands' patients to be of rheumatoid origin, he advanced as an alternative to Dr. Rowlands' deductions that the intestinal toxæmia and the bacilluria were the result of, or coincident with, but not the cause of, the arthritis.

In the light of this explanation, together with the fact that no controls of the vitamin B therapy against the bacilluria were recorded, he failed to find wherein Dr. Rowlands was justified in correlating his clinical with his experimental experiences.

Dr. J. F. HALLS DALLY said that thus far little direct evidence of pathological changes in the joints during a period of vitamin-B deficiency had been adduced. The crux of the matter appeared to be this: If, in the joints of experimental animals subjected to a definitely devised vitamin shortage or deprivation, changes were found which, macroscopically and histologically, corresponded with those which were universally acknowledged to occur in rheumatoid arthritis, a probable causal association of a certain type or types of vitamin shortage or deprivation with rheumatoid arthritis would have been established.

Dr. Rowlands had told him that joint-swellings had been observed in certain of the animals, but that the time interval required for the production of further changes in the joint tissues rendered the investigation difficult, and a more extended inquiry was therefore necessary.

He (the speaker) submitted that Professor Plimmer's use of the term "deficiency," to indicate absence of vitamin in a diet, was likely to lead to confusion. Deficiency meant inadequacy, i.e., not having a full supply, and was equivalent to shortage. If *complete* absence was meant, would it not be better to use the term "deprivation"?

During the past two centuries a systemic disposition to particular diseases or "diathesis" had been recognized by physicians. This conception, however, became swamped by discoveries in bacteriology and pathology, and "diatheses" went out of fashion, only to appear later in modern biochemical and bioclimatic form as the expression of an individual lack of immunity consequent upon some metabolic defect or excess. Thus "constitution" can now be assessed in terms of biochemistry, and "diathesis" holds a wider, though more precise, significance than it did in the eighteenth century.

Of diatheses, the "rheumatic" was one upon which increasing attention was being directed. From the essential unity of the pathological joint changes which occurred in acute and chronic forms of rheumatism, blending into or alternating with one another as they did under the influences of age and constitution, it would appear reasonable to predicate a unity of origin, and he (the speaker) would suggest that such was to be looked for in errors of metabolism. The first group might be called "endocrine."

I. With the onset of puberty and the endocrine adjustments necessitated by the advent of gonadic activity, rheumatic symptoms of childhood blended with those of adolescence. In the adult, acute muscular might alternate with acute articular rheumatism, and in rheumatoid arthritis Coates and Gordon had shown that the frequency of antecedent rheumatic fever was greater than could be accounted for by its general incidence in the population. Llewellyn, too, had pointed out that, like acute rheumatism, rheumatoid arthritis was linked with states of hypo- and hyperthyroidism and with deficiency of ovarian and pancreatic secretions. It was becoming appreciated that administration of various hormones, especially of small doses of thyroid and parathyroid glands, produced definite benefit, whilst withdrawal was followed by recession of the disease. So-called "climacteric" arthritis was of this type, but, since the clinical

66 Rowlands: *Rheumatoid Arthritis: Is it a Deficiency Disease?*

manifestations were not peculiar to the menopause, in a recent discussion at the Section of Balneology[1] the speaker had advocated the term "endocrine arthritis" as being in accord with what he believed to be the true ætiology. Links between the more defined conditions were to be found in the occurrence of tetany, migraine and Raynaud's disease, and a considerable mass of evidence had now accumulated which went to show that the underlying element for a large group of rheumatic disorders which manifested a failure of adaptation to environment, climatic and general, as a result of lowered metabolism, was to be found in a condition of subthyroidism with suboxidation.

The suggestion was, therefore, that the endocrine and trophic defects, already referred to, with the associated intestinal atrophy and stasis, originated from a basis of malnutrition caused by a diet insufficient either in quantity, or quality, or both, particularly as to the content of particular vitamins. An interesting sidelight on Dr. Pulvertaft's remarks was afforded by the recent emphatic statement of Dr. Arbour Stephens that bad feeding was the chief disposing cause of heart disease in children, or in other words, the heart disease was a deficiency disease due to the lack of food containing lime as well as vitamins. This absolute statement might need some qualification, but in the main it accorded with the views expressed as to changes in cardiac nutrition found after vitamin-B deficiency.

II. Another group, also of metabolic origin, was in essence biochemical, arising from inherent defect in capacity for assimilation, even if the diet was sufficient both in quantity and quality. This group, according to Dr. Henry Ellis, was associated with a diminished urinary excretion of phosphoric acid. Such patients exhibited an "alkaline" type of constitution, with small hearts, diminished peripheral tone and consequent hypopiesis, both systolic and diastolic, which seldom attained normal limits. The differential pressure being low, these patients had a sluggish circulation with cold extremities, general lowering of vitality, intestinal stasis and toxæmia.

III. The third type of metabolic error was opposite to the former two. It was either constitutional or acquired, and consisted in retention of waste katabolic products by reason of faulty elimination. These subjects were of the "acid" diathesis, and tended to develop osteo-arthritis, frequently monarticular and hypertrophic, in contradistinction to the two former types of multiple and atrophic rheumatoid arthritis. Other important contrasts were found in the association of high arterial pressures, arterio-sclerosis and large hearts.

Finally with regard to the question, "Is rheumatoid arthritis a deficiency disease?" he (Dr. Halls Dally) was of opinion that vitamin deficiency would probably be found to be only one of several factors concerned in the ætiology of rheumatoid arthritis.

Sir WILLIAM ARBUTHNOT LANE said he had devoted much attention to the study and treatment of rheumatoid arthritis and was familiar with its various clinical manifestations. Long ago he had realized the fact that rheumatoid arthritis, like many other diseases, resulted from a lowered resistance due to infection of the food contents in the small intestine by organisms which had invaded it from the large bowel. This ascent of organisms from the large bowel was produced by an accumulation and stagnation of fæcal matter in it; in other words, it was the result of habitual constipation.

Dr. H. A. ELLIS said that though vitamin deficiency might sometimes be an activating cause of rheumatism the primary constitutional condition was biochemical. There were three types of constitution: (1) That in which intake and output were balanced (normal); (2) that with insufficient intake (assimilation error); (3) that with restricted capacity of output due to incompetence in getting rid of waste (elimination error).

Rheumatism did not often attack the normal type, but was usually confined to the other two groups, in both of which there was a tendency to deficiency of phosphoric acid as shown by urinary analyses. This was the more remarkable because in the elimination error cases there was an excess of acidity as well as an excessive specific gravity until the kidneys began to fail. In the assimilation error group also, this phosphoric acid deficiency was out of proportion even to the general deficiency. The incidence of rheumatic symptoms in each class was quite distinct; in the assimilation error type they usually began with the small joints (rheumatoid arthritis); in the elimination error cases they were usually connected with the larger joints (osteo-arthritis). The group of cases described as the climacteric variety, activated by thyroid deficiency, largely belonged to this second class.

[Tables were shown giving the urinary analyses of (1) an average normal individual, (2) a patient suffering from typical osteo-arthritis with excess ratio of output, (3) a patient

[1] *Proceedings*, 1927, xx (Sect. Baln.), 12.

suffering from arthritis deformans with deficiency of intake. In (2) and (3) there was a deficiency of phosphates.]

In dealing with the deficiency of vitamin B in cases of rheumatism, Dr. Rowlands' paper was convincing and dramatic, but the relationship between this deficiency and the various forms of rheumatism was not clearly shown. Whereas it was probably a factor in rheumatoid arthritis, the co-relation was not evident in either osteo-arthritis, with its prevailing characteristic of robustness, or in the climacteric type associated with thyroid deficiency. Possibly there were other vitamin deficiencies—an "A" deficiency and probably a "D" deficiency—concerned in the control of phosphates. As to the quantity of "B" required, he doubted the wisdom of relying on tests made on the pigeon, since temperature, pulse-rate and superficies differed from those of human beings, and all these entered into metabolic calculations.

With regard to giving "B," he (Dr. Ellis) had been so strongly impressed with the neurotic side of the disease that he had been giving "marmite" for over eighteen months because of its vitamin content, but had not obtained any of the desired results. His hope in giving vitamin D was that it might control the phosphate metabolism.

Dr. Rowlands' view, however, that vitamin-B deficiency was an essential factor, was in close accord to his (the speaker's) clinical observations.

He did not consider arthritis deformans to be a joint disease at all, but to be a trophic and autonomic nervous paralysis showing itself in wasting and loss of tone of the muscles before any joint injury supervened. The muscles were under constant tonic irritation and degeneralization, and, as always in such paralytic conditions, the flexor muscle won. The hands of the rheumatoid arthritic always showed an identical deformity; they tended towards flexure and eversion. The wrists were dislocated forwards, and the knees flexed backwards, and in all cases the deformity was always similar. That such a condition was the result of a joint disease was unthinkable.

He had a case at present of rheumatoid arthritis in which only a few of the small joints of the hands were affected, but in which every muscle of the body was wasted beyond repair. The disappearance of the interossia and the flattening of the back of the wrist occurred before the joint lesions, and there were two conclusive symptoms that the condition was not joint disease but a trophic paralysis. One was the "feel" of the skin of the palm of the hand, which was like velvet and varied with the condition of the patient; the other was the face of the individual, which, despite the agony suffered for years, had no line or appearance of pain or stress. This was due to the facial paralysis which always existed and which in this case could not be associated with any joint. It was interesting to see the return of the lines of pain as the case improved.

Rheumatoid arthritis was certainly a deficiency disease, and the deficiency was connected with the assimilation or utilization of phosphoric acid and other phosphates, so that probably vitamins B and D were often associated with it. Rheumatoid arthritis never attacked the *bon viveur* or the alcoholic, but was the disease of the total abstainer, the vegetarian and the careful liver.

Dr. CAWADIAS said he could not agree that rheumatoid arthritis was a deficiency disease. Deficiency, in any case, could only be considered as one of the multiple external causes, and in rheumatoid arthritis the external causes were quite secondary, the principal ætiological element being the constitutional predisposition.

Dr. ROWLANDS (in reply) said that most of the speakers had evaded the real point.

He had been asked what he meant by rheumatoid arthritis, but he would prefer to leave the definition of it to his audience; or as it had been given by Dr. Llewellyn in his book, or in Dr. Hare's paper in the *Veterinary Record*. The experience of Sir Arbuthnot Lane, as one of our well-known surgeons, must carry weight, as it did with the speaker.

He (Dr. Rowlands) had not begun this work with the object of proving that rheumatoid arthritis was a deficiency disease. What he had tried to discover was the cause of the intestinal stasis, so prominent a symptom in every case of rheumatoid arthritis under his care. A large number of the patients were constantly being examined by X-rays by Dr. Finzi, and in many of them the appendix did not fill, and the food was late in getting through the intestine; there was a delay of from twenty-four to forty-eight hours in the "deficiency" animals in comparison with the normal. When he and his colleagues came to the feeding to these cases of streptococci, staphylococci and diphtheroid rods, they hit upon something else, which he had detailed in the paper. Why was it that in his cases of rheumatoid arthritis

68 Rowlands: *Rheumatoid Arthritis: Is it a Deficiency Disease?*

89 per cent. were infected with the *Bacillus coli*, 6 per cent. had streptococcus, and 1 per cent. the bacillus of typhoid? He had seen ten new cases in the past week, all of which grew the *Bacillus coli*.

Dr. Vincent Coates had discussed infective arthritis, but he (Dr. Rowlands) was not dealing with that at all. He agreed with Dr. Coates that a disease like influenza, also worry, would predispose to any condition. People who were overworked and worried were liable to get any infection, because their resistance had thereby been lowered. Dr. Coates agreed there was wasting of the muscles.

Dr. Ellis, towards the end of his remarks, had said that he regarded rheumatoid arthritis as a nerve disease. That was the whole secret of the investigation.

Dr. Cramer had asked the speaker whether he was certain he was not dealing with vitamin-A deficiency. The answer to that was simple. If he put the animals on a diet containing sufficient vitamin B they recovered.

Several speakers asked what was the condition of the joints in these animals, and the remarks of Dr. Halls Dally helped to supply the answer.

Several speakers made remarks with which he could not agree, but there was not time to controvert the points raised. Dr. Ellis said he had given marmite to his patients, but without benefit. He (Dr. Rowlands) was not surprised at that—in his own cases he had not given marmite; he had given the wheat embryo. If Dr. Ellis would turn his attention to the wheat embryo he would obtain the same results in his cases as he (the speaker) had obtained. Possibly biochemists would soon discover what was in the wheat embryo which was not in marmite or yeast extracts.

The patients certainly improved when treated on the lines that he had suggested.

He was sure that endocrine pathology—thyroid-testicular-ovarian—was concerned in rheumatoid arthritis, but his contention was that the endocrine balance was overthrown owing to the deficiency. The patients were getting a certain amount of vitamin B in their diet, but not enough. A rat would live on a small quantity of vitamin B for two or three years, but if such a rat were killed in the eighteenth week of experiment, it was found that there was scarcely any muscular tissue remaining; the external muscular coat had gone, and the internal muscle was only one-third the normal size. And in every deficiency animal, as Dr. Browning had said, there was a practical absence of peristaltic wave. Yet in a normal animal, if it were killed with chloroform and opened and exposed to bright sunlight, the peristalsis persisted for twenty minutes. Certainly in most cases of rheumatoid arthritis there was constipation; in some there was possibly colitis, since there were repeated motions.

A question which could not be evaded was: Why was the *Bacillus coli* found in the urine in so many rheumatoid arthritis cases? It could only come from the intestinal canal, and it was not able to penetrate healthy mucous membrane. In the animals and in the human patients in these cases, the healthy mucous membrane had disappeared as a result of vitamin deficiency.

An important point which none of the discussers had mentioned was the great change in our diet, not so much in our own choice of food, but in the food of the animals on which we depended so much for our own. For instance, cows used to be fed on ground oats, ground wheat, ground barley, ground rye; all these contained the essential vitamin B. To-day very few farmers gave such food to their cattle; instead, they gave cotton-seed cake, linseed cake, and all kinds of patent foods which were deficient in vitamin B, and therefore milk was not now so good as in former days. Chickens, again, were now fed on all sorts of material, and were the subjects of intensive culture, with the result that the egg-yolk was not of the same value as formerly. Vitamin B was not an animal product, it must be supplied to the animal from some outside source.

He (Dr. Rowlands) agreed with Dr. Halls Dally that rheumatoid arthritis was a nerve disease following on toxæmia.

To ascertain whether joint lesions would occur in the experimental conditions, it would be necessary to go a step further. He would like to keep experimental animals for six months, but he would not be allowed to use the pigs on his farm as experimental animals, and University College could not be turned into a row of pig-styes.

He had learned something from the discussion. He was extremely obliged to the speakers for the kind way in which they had received his paper. He knew he was treading on very thin ice, but he was advised to place his findings before the Section, and he hoped the resulting discussion would go further. Probably a treatment would be instituted on the right lines, and thus justify his thesis.

Section of Laryngology and Section of Otology.

Chairman—Dr. ANDREW WYLIE (President of the Section of Laryngology).

COMBINED SUMMER MEETING.

LARYNGOLOGICAL SESSION.

Chronic Hyperplasia of the Upper Jaw.

By F. BRAYSHAW GILHESPY, M.R.C.S., D.L.O., F.R.F.P.S.Glas.

I HAVE been prompted to communicate this paper for the following reasons:—(I) To recall attention to the salient features in Westmacott's original article on the subject in 1913; (II) to point out the efforts made to include these cases under the wider heading of osteitis fibrosa; and (III) to indicate certain similarities between this disease and otosclerosis.

In the eight cases recorded by Westmacott, the patients, whose ages ranged from 17 to 26 when the condition was first noted, suffered from a bony unilateral enlargement of the upper jaw. It is interesting to note that in six of the cases septic teeth were present in the affected portion; in one the upper jaw was edentulous. At operation the new bone could be gouged away, being of moderately soft consistence. Sections of the portions of bone removed showed a remarkable similarity in the changes present. The report on the second case, which may be taken as an example, is as follows:—"The tissue consists of well-formed bony tissue. The laminæ of bone are already laid down and the bone corpuscles stain well. The interstices of the bone are occupied by a thick layer of actively proliferating osteoblasts, amongst which are a few osteoclasts. In addition there is a rich vascular supply and an abundance of connective tissue cells; these cells possess an unusually large amount of protoplasm. There are no collections of small cells or polynuclear leucocytes, nor are there any signs of granulomatous inflammation. The condition appears to be one of simple hyperplasia."

From these findings Westmacott concluded that there was a reversion of the bone to the embryonic type which is present in the upper jaw before the formation of the maxillary antrum, and of which remains are found in the adult, only in the alveolus and malar process. He pointed out that the premaxilla was unaffected in all cases, and considered that this fact proved that the condition began in the alveolus. Infection from the nasal accessory sinuses has never been suggested as a cause of the changes in the bone, but dental sepsis, as a factor, must be considered, since it is present in the majority of recorded cases.

Douglas Guthrie, at Manchester, in 1923, mentioned two cases under his care. Microscopic sections showed numerous trabeculæ of new bone with a reticular arrangement and the meshes of this reticulum were occupied by a loose, fibrous tissue. In these sections very little evidence of inflammatory reaction was present.

Lawford Knaggs, in 1923, wrote a full description of "Leontiasis Ossea," a title which had been used to designate those hyperostoses of the skull the nature of which had not been understood. He made a division of this disease into two varieties; the first taking the form of a slowly spreading periostitis, the second, of a diffuse osteitis. "Creeping periostitis of the bones of the face and skull" is a very descriptive name for the first variety, which seems to arise in the nasal fossa or the sinuses and is probably due to infection in these regions. Beginning in the

76 Gilhespy: *Chronic Hyperplasia of the Upper Jaw*

periosteum of the sinuses, the infection creeps beneath that membrane—at the same time causing an osteitis of the underlying bony tissue—and emerges into the nasal fossæ, travelling thence to the outer surface of the maxilla or spreading backwards into the pterygomaxillary fossa to the posterior surface of the superior maxilla and into the temporal fossa. The two streams eventually meet. The spread of practically normal new bone under the periosteum is relentless, causing the characteristic bossings and obliterating the sinuses affected. The records of such cases are deficient in rhinological reports, as Kelson has pointed out. The disease is admittedly rare, but we have the opportunity and responsibility of diagnosis and treatment in the early stages. It would be of great interest if any Member could give us details of a case seen over a long period, and whether treatment of the sinuses has in any way mitigated the severe course of the disease.

In the second class—that of diffuse osteitis of the bones of the face and skull—the affected bone in the early stage is soft and vascular, the cranial bones may be remarkably thickened and the inner and outer tables cannot be differentiated, the structure of the bone being homogeneous. The age of onset of this condition is usually in the first or second decade of life. These changes may be general in the cranial and facial bones, or may affect only one or more adjacent bones. This class is of interest to our Section, and cases have been shown by Members. I have under my care a patient whose left superior maxilla was removed ten years ago on account of a growth supposed to be sarcoma. The patient now has a hard extension of growth towards the frontal region on the same side, but X-rays reveal a general thickening of the base of the skull, which compels revision of the original diagnosis of sarcoma. Knaggs puts cases of this kind into the category of osteitis fibrosa, and also considers those of chronic hyperplasia of the upper jaw to be cases of osteitis fibrosa. Cases shown at this Section have demonstrated that sometimes the enlargement of the upper jaw is made up of hard bone, without much trace of fibrous tissue, a condition not found in osteitis fibrosa occurring in long bones. Knaggs recognizes this and explains how it might be related to the difference between the blood-supply in a skull bone and that in a long bone supplied by a nutrient artery.

In a case under my care at present the report on a section is as follows:—

"The specimen consists of cancellous bone with extensive deposition of fibrous tissue in the interstices. Osteoclasts occur irregularly through this tissue. In places it is fairly vascular, elsewhere it is practically cicatricial in character. Some of the bony laminæ are well formed and the bone corpuscles stain clearly; in other parts the bone has a degenerated appearance; the corpuscles have almost lost their power of staining and the bone is undergoing absorption. Nothing suggestive of any inflammatory or granulomatous process is seen in any of the sections.

"HASWELL WILSON, Professor of Pathology."

My patient was a woman, and this disease appears to be more common in females. The enlargement of the upper jaw could be easily gouged away. The teeth were septic on the affected side; the antrum was neither obliterated nor infected. From the description of my sections it would appear that we are not dealing with a hyperplasia of bone but rather with an osteoporotic process. Recently Harper showed a case in Scotland, which some members of the Scottish Laryngological Society considered to be one of chronic hyperplasia; but the exhibitor had entitled it a fibroma.

Can we account for these changes found in the superior maxilla? Rehn has described an affection of the facial and skull bones in pigs which he has related to an inflammatory septic process starting from the teeth, but his proofs that the infection is the basis of the bone changes are not admitted by pathologists generally

Cases recorded have generally been seen in an advanced stage, and it is possible that the changes in the teeth may be secondary.

A case was recently shown by Mr. Howarth, in which there were changes, seen by X-ray examination, in the basi-sphenoid and vault of the skull, demonstrating that the disease in this case was not localized to the superior maxilla. Dawson and Struthers have compared the changes found in otosclerosis and in chronic hyperplasia of the upper jaw. They point out that in both conditions changes are found in localized areas of bone which bear a striking resemblance to those described in osteitis fibrosa. In both conditions there is a close relationship to a mucous membrane, frequently the site of septic infection. In conclusion: Are we to consider chronic hyperplasia as a simple fibrous osteitis (as seen in the bone near an infected bunion) and arising from dental infection, or is it a localized dystrophy of bone, in the nature of an osteitis fibrosa depending on a biological variation incited to activity by various possible causes, of which infection from the mouth or teeth may be one?

Discussion.—Mr. WALTER HOWARTH said he had recently shown two or three cases of this kind which he had followed up for several years. In one of these he had removed the swelling seven years before and in another three years before, and the condition afterwards had remained stationary up to the present time. He did not suppose that he had removed all the disease. In both those cases the original site was the alveolus, and most probably dental sepsis was the real cause.

[Mr. HOWARTH showed a specimen of an upper jaw from a patient who had fallen dead in the ward from heart disease.]

Dr. W. H. KELSON said that he had only had two such cases under his care, and in neither was there any dental sepsis; clearly extensive removal of teeth was not warranted. Horsley had had considerable experience of these cases, and was inclined to operate freely; but after free operation the patients seemed to go from bad to worse. There was need for a closer study of the pathology of this rare condition.

Dr. T. RITCHIE RODGER said he had read a paper before the Scottish Laryngological Society describing a case of the kind, which he had entitled leontiasis ossium. There had been large bosses on the left superior maxilla, and one on the right mandible. He had lost sight of the patient for three or four years; then she had returned and several bony rhinoliths had to be removed. The Wassermann reaction, which had previously been negative, was then positive. He (the speaker) advised that this test should be carried out repeatedly in these cases.

Mr. DOUGLAS GUTHRIE said that in the Museum of the Royal College of Surgeons there were two specimens of hyperplastic upper jaws which had been removed, presumably because the operator thought he was dealing with malignant growth. He (the speaker) showed slides of these bones illustrating how the antrum became compressed and small. One of the specimens had been removed by Lord Lister; in no case was there associated dental sepsis or decay.

He also showed the photograph of a small boy whom he saw six years ago, the condition having been verified microscopically. He saw the boy again recently, and the size of the swelling of the jaw had not increased. The condition apparently caused no inconvenience.

Another case, that of a girl aged 15, he had followed for several years. Recently tuberculosis had developed and the patient was now in a sanatorium. In this case there was definite dental sepsis.

Mr. J. HARPER said he now had three cases of the kind under his care; one in a girl, aged 15, with extensive enlargement of the right upper jaw. He first saw her four years ago, when he chiselled away as much of the bone as he thought necessary in order to improve the contour of the face. A month ago she returned with a projection upwards of the floor of the orbit. Microscopic sections showed the usual fibrous changes, but no malignancy. It had now become necessary to remove the jaw in order to save the sight.

78 Howarth: *Some Points in the Technique of Laryngo-fissure*

Dr. J. S. FRASER said that the appearances shown in Mr. Gilhespy's lantern slides were much like those seen in the labyrinth capsule in otosclerosis, and pointed to both conditions being of a chronic inflammatory nature. There was some support for his view that otosclerosis was a chronic inflammatory process. The bony inner wall of the middle ear was covered by mucous membrane, and the inflammatory process might spread from the deep layer of the mucous membrane into the bone in the region of the anterior margin of the oval window. There were special circumstances in this region which rendered infection liable to occur, such as the anastomosis between the vessels of the labyrinth capsule and those of the periosteal bone, as described by Politzer. Another factor was the presence of the tensor tympani tendon.

Dr. LOGAN TURNER asked that this subject might be taken up as a collective investigation in connexion with the general pathologist, and that a small committee might be appointed.

Mr. GILHESPY (in reply) said that interesting cases of osteitis deformans had been shown recently by Mr. Jenkins, and these cases bore an analogy to those of otosclerosis, as Dr. Fraser had pointed out.

Some Points in the Technique of Laryngo-fissure.

By WALTER HOWARTH, F.R.C.S.

IN the surgery of malignant disease it is, I believe, the most profound mistake to be tied to a set operation. The operation of laryngo-fissure for cancer of the vocal cord is, however, in some ways an exception, as not only are we able to define accurately the limit of the growth by both indirect and direct methods, but, if by any chance the case should turn out to be unsuitable for this procedure, it is comparatively easy to switch over to a more extensive operation. The operation of laryngo-fissure has gradually been perfected and a definite technique established. Many of us in this room have been fortunate enough to be allowed to watch the various steps of the operation unfolded by the hand of a master, and not one of us but has learnt some valuable lesson each time. Everyone, however, must eventually work out his own salvation, and in the process every thinking surgeon must inevitably explore possible avenues of simplification in the perfection of his technique. All progress comes by experiment, but experiment is only justifiable if it is entirely consistent with the safety of the patient.

In considering this operation the points that I wish to bring before you are: (1) The necessity for tracheotomy; (2) the advisability of removing the thyroid ala; (3) the possibility of diathermy.

Preliminary tracheotomy has, I think, many disadvantages. Not only does it add an operation with its attendant surgical shock, but the open trachea, however carefully shut off with plugs, may allow blood and secretions to pass into the lungs. The administration of the anæsthetic through the tracheal opening is apt to interfere mechanically with the comfort of the surgeon, and the direct stimulation of the tracheal mucosa does not always make for smooth anæsthesia. Moreover, some patients tolerate tracheotomy badly, probably—as pointed out by Mr. Negus—owing to the loss of the movements at the glottis disturbing the "sympathetic connexion" between respiration and circulation. I recall at least one case in which tracheotomy was probably the last straw in the balance against a fatty heart.

I have for a long time tried to banish the anæsthetist from the site of operation, and fifteen years ago I showed before this Section a case in which laryngo-fissure had successfully been performed under infusion anæsthesia with hedonal, the anæsthetic being introduced through the veins of the foot. This method was unfortunately given up owing to some fatalities occurring after its use in unsuitable cases and in unskilled hands. The method I now advocate is the intratracheal administration of ether without tracheotomy. The catheter lies in the interarytænoid space, and, when

the larynx is opened, is scarcely visible and not in the least in the way. The anæsthesia is smooth and there is no danger of any blood getting into the lungs: two facts which ensure tranquillity in operating and enable the surgeon to devote his whole mind to the removal of the malignant growth and the immediate arrest of any bleeding, without anxiety lest blood should get into the lungs, and the constant mopping—to prevent its doing so—which not only damages the laryngeal mucosa but may implant cancer cells from the growth. It may be said that the absence of a tracheal opening omits a safeguard in the event of post-operative hæmorrhage. I question whether a tracheal opening is really a safeguard. If some slight oozing should occur, it is in any case got rid of by the mouth. If the bleeding is profuse and demands local measures, it is the work of a moment to slip a laryngotomy tube into the lower end of the wound and pack the larynx above this. In cases of hæmorrhage everything depends on skilled help being immediately available, and we should hear little of fatal cases due to bleeding if the surgeon were close at hand for the first twenty-four hours. If the larynx is completely closed without a tracheotomy, there is one other incident that may happen, namely, the occurrence of surgical emphysema of the neck and face. This can readily be prevented by inserting a very thin piece of rubber tissue into the lower end of the wound and keeping it there for the first twelve hours.

(2) We now turn to the question of the removal of the thyroid ala. This was suggested, in 1916, on the grounds that it ensures more thorough removal of the disease and enables one to control the bleeding better. It is also maintained that there is no subsequent necrosis of a fragment of cartilage, that there is no post-operative difficulty in swallowing, and that the after-results of the voice are excellent. I have followed this procedure for many years, but have now given it up and returned to the previous method of removing the tissues within the ala. If the malignant growth is situated on the vocal cord (and few cases except those so situated are really suitable for laryngo-fissure), one can be perfectly certain of being very wide of the growth without removing the cartilage barrier, whilst in the unlikely event of a recurrence taking place, this will probably occur within the cartilaginous framework instead of being diffused through the tissues of the neck. The recurrence is more likely to be recognized and can be dealt with by laryngectomy or other methods, whereas previous sacrifice of the laryngeal structure makes early recognition of recurrence difficult and a further operation a matter of the greatest uncertainty. It may be said that malignant growths in the larynx tend to spread in the subglottic space and to escape externally through the lateral part of the crico-thyroid membrane, and that removal of the ala enables the surgeon to circumvent this extension. I wonder whether much is really gained in this way—but be that as it may, growths that extend into the subglottic space are those that are most likely to recur, and are not to my mind suitable for laryngo-fissure.

With regard to the other points, necrosis of a fragment of the thyroid ala can occur, as happened in one of my own cases, and may cause a very troublesome local abscess situated deeply at the side of the larynx. Difficulty in swallowing is not due to any wrenching apart of the alæ of the thyroid, as alleged, but is invariably due to interference with the arytænoid. This should not happen. The after-results as regards the voice are not, in my experience, nearly so good as when the thyroid ala is left intact.

(3) Diathermy is, as yet, a comparatively untried procedure in this operation, and I myself have only employed it to stop bleeding in a case in which the vocal process was removed and bleeding from the artery in this neighbourhood was exceedingly troublesome. The result was most satisfactory, and I shall now regard diathermy as likely to prove valuable in checking persistent oozing and, moreover, to do

80 Howarth: *Some Points in the Technique of Laryngo-fissure*

considerably less damage than the endeavour to apply pressure forceps when there is no bleeding point obvious. I have not used the diathermic knife for the complete removal of a vocal cord, but this procedure might well be tried and should ensure a rapid and bloodless operation with a resultant scarring more resilient than that usually obtained. If diathermy be employed, naturally ether is inadmissible as the anæsthetic, but it only takes two or three minutes to wash out the lungs with oxygen, and then chloroform may be given through the intratracheal catheter. If a complete diathermic removal is contemplated, it would probably be better to use intratracheal chloroform from the beginning.

To conclude: The operation of laryngo-fissure is eminently suited for malignant growths of the true vocal cord, but in the performance of this a tracheotomy is not necessary if intratracheal ether is used. Nor does the removal of the thyroid ala seem to be advisable. The possibility of using diathermy is one that might well be considered, not only for the stopping of troublesome oozing, but also for the actual removal of the cord.

Discussion.—Dr. ANDREW WYLIE (Chairman) said that in two cases in which he (the speaker) had performed laryngo-fissure without preliminary tracheotomy, he had had difficulty with the hæmorrhage, and after the operations the patients had coughed so severely that he was obliged to open the trachea immediately.

Professor G. PORTMANN (Bordeaux) said that in his clinic the custom was to do this operation without preliminary tracheotomy. If there was severe hæmorrhage—but that was exceptional—one was not in a good position to deal with it, but one tracheotomy could then be performed. The rule was to perform the operation under local anæsthesia, and usually there was no trouble.

Mr. N. S. CARRUTHERS said he questioned whether no blood at all passed down the trachea when ether was administered intratracheally.

The tube lay in the posterior part of the trachea, and at the point of contact the upward pressure of air was insufficient to prevent blood flowing downwards. This fact could be easily demonstrated in the laboratory. The small quantity of blood so passing might account for the severe bronchitis or pneumonia which occurred in some of the cases afterwards.

Sir STCLAIR THOMSON said that, having himself performed some eighty laryngo-fissures, he had naturally arrived at certain opinions. But while holding certain opinions, it was very important for the surgeon's outlook that his opinions should not hold him. He therefore tried to rid his mind of prejudice—a very difficult matter.

With regard to the question of the removal of the thyroid ala. He had seen the beginning of laryngo-fissure in this country, as he had helped Sir Henry Butlin and Sir Felix Semon in that operation, and they did not remove the cartilage in question. Its removal, he thought, was first suggested by Broeckaert, of Ghent, at a congress in Paris, some time before Dr. Lack suggested it. Besides, Dr. Lack did not suggest it as a step in the median laryngo-fissure operation, but as a window resection from the side. He (Sir StClair) first tried it on Broeckaert's suggestion. Before employing it he had had two or three cases in which there was some necrosis, and small portions of the cartilage were coughed up during convalescence. Since he made a practice of removing the ala that had not happened. That the ala might form a barrier was a very wise suggestion, and in the study of malignant disease this question of Nature's barriers and how they could be conserved was one which merited careful study. In one of his early cases, in which he had not removed the cartilage, the patient had returned seven years later with an epithelioma on the opposite side, and a second laryngo-fissure was done; he had shown that patient recently (four years after the second operation) to the American surgeons who were visiting this country. There had been no recurrence, a fact that might be due to his having left the cartilage, which had formed a barrier. He had, however, had another case, that of a female, aged 33, in which he was doubtful whether the laryngo-fissure would be successful, as the whole extent of the cord was involved and it did not move very freely. However, he had performed the operation and removed the ala. Within five months there was a recurrence, but the patient deferred coming for another three months,

and when she returned the whole side of the larynx was so fixed and infiltrated with the growth that he had handed her over to Mr. Wilfred Trotter, who had operated successfully and turned in a flap. That was seven years ago, and the patient was still well and earning her own living. She wore a tracheotomy tube because of the stenosis. Removal of the ala gave a larger field. It was a mistake to talk of the alæ being forced apart. They could be fairly well separated, and if one ala was taken away and only soft tissue was left, there was a freer approach, and one could study the extent of the trouble. In the cases in which the ala was removed the glottis was larger than in those in which it was left, and the bare cartilage on one side was more slowly covered over with granulations than were soft tissues. The longer that covering process took the greater was the contraction, and in the cases in which he had left the bare ala to cicatrize over there was a smaller glottis than when he had removed it. He could show four cases of laryngo-fissure in which the patients were medical men who were now using their voices, the most recent operation being two years ago. The barrier did not consist in the cartilage, but in the perichondrium, and he was always careful to preserve the external perichondrium.

With regard to preliminary tracheotomy, his (Sir StClair's) principle was "safety first." Tracheotomy added no danger to the operation, and it caused no shock. He had operated on a patient aged 80, whose blood-pressure was 200 mm., and had had occasional attacks of angina pectoris. That patient had been sitting up and playing cards within twenty-four hours of the operation, and was alive and well three years afterwards. Another patient, with a blood-pressure of 200, was out of bed reading on the evening after the operation. The injection of cocaine, and other precautions, prevented coughing, splashing, loss of blood and shock. He could not understand how a fairly large ether tube in the interior of the glottis could facilitate the view of the extent of the disease. Blood could be cut off absolutely by tracheotomy and the careful packing with gauze above it.

Reports of successes along various lines were published from time to time, but little was heard—except indirectly—of failures and of the accidents which occurred owing to the non-insertion of a tracheotomy tube—three of these had come to his notice during the previous winter. He (Sir StClair) was anxious that the high reputation which laryngologists in this country had obtained with regard to this operation should be maintained. In his (the speaker's) eighty cases of laryngo-fissure there had been, as everyone knew, three deaths; one of those was due to rupture of the œsophagus, and the other two followed mistaken doses of heroin and morphia.

Mr. W. M. MOLLISON said he had performed laryngo-fissure on two or three occasions with intratracheal ether, and the tube had not been in his way; it lay in the posterior commissure, out of the line of vision.

Mr. HOWARTH (in reply) said that in his later cases he had had better results and an easier passage than in earlier ones in which he had first performed tracheotomy. In his early laryngo-fissure cases he had had three deaths, and those were caused by blood getting into the trachea. He had not found happen what Mr. Carruthers mentioned. There was always a return current of air which blew anything upwards, even from the upper end of the trachea. He thought that if the ala was removed there might be a more diffuse recurrence in the neck tissues. He did not think the ala greatly limited the view.

The Relation of Nasal Polypi to Inflammation of Accessory Sinuses of the Nose.

By T. B. LAYTON, D.S.O., M.S.

POLYPI were at one time classed as innocent tumours [1] and were labelled "myxomata." We are now agreed that they are inflammatory; but of their meaning and of their exact nature we are still ignorant. Are they, for instance, indicative of some special form of inflammation of the mucous membrane? or are they the secondary results of some other disorder? In the former case, they themselves constitute the disease and to treat them by removal may be right; if the latter, they

are merely objective symptoms, and to remove them can do no good except temporarily to relieve the subjective symptom of obstruction.

Do polypi ever exist without any underlying disease of the accessory sinuses? Some authorities say, yes [2], others, no [3]. The more I search for some disease of the accessory sinuses in persons having nasal polypi, the more often I find it. I believe that the only occasions where there is none occur when polypi are present in a very roomy nose, and I suspect that in such persons the polypi are rather of the nature of hypertrophies compensatory to the excessive airway than that they are inflammatory structures.

Does removal by snare or forceps ever cure polypi? In my experience, very seldom, and then only when these are single and have a stalk arising beneath the middle turbinal. The removal of multiple polypi is so disappointing in its results that I have almost given it up, unless, for age or other reason, more radical treatment cannot be done.

What type of inflammation of the sinuses leads to polypi? To answer this question let us ask another. What are the varieties of sinusitis? and let us accept Hajek's classification [4] as a basis of discussion. He describes: (1) Acute catarrhal; (2) acute suppurative; (3) chronic catarrhal; (4) chronic suppurative; and (5) diphtheritic. The last we will rule out. It must be very rare, and I have never seen it. I hold with Hirsch [5] that the two chronic forms are separate and distinct lesions, and that polypi are associated with the catarrhal form and not with the suppurative. Let us be quite clear what is meant by two separate and distinct lesions. I mean that they arise in different ways, run separate courses and do not pass the one into the other. Two conclusions follow: (1) That polypi do not accompany suppurative sinusitis; and (2) that chronic catarrhal sinusitis is not a result of suppurative sinusitis, either acute or chronic.

In a case of polypi I used first to remove them, and when the wounds were healed whole, the nose was clear and before the polypi had had time to form again I used to search for signs of suppurative sinusitis. As my intranasal technique improved, and as I became more sternly self-critical of every stage of my examination I was unable to find evidence of it. It may be asked: "What of those cases in which streams of muco-pus are seen within the nose amongst the polypi?" Usually this muco-pus comes from the nasal fossa itself, and if sufficient time be spent in making sure that all is removed, there is not that return within the half to one hour upon which the diagnosis of suppurative sinusitis is made.

Sometimes a small amount of pus can be obtained from an antrum affected with a chronic catarrhal inflammation. This, however, differs from that seen in suppurative sinusitis, in the smallness of its quantity, in its inconstancy from day to day, and in its consistency; it is thick, tenacious muco-pus, not the liquid pus or inspissated mass seen in suppurative inflammation. Either it is due to a suppurative affection grafted on to the catarrhal, or, as I suspect, it is muco-pus escaped into the antrum from the nose. Sometimes, however, true pus, which returns after being mopped away, is seen in a case of polypi; in such a case I have found that one sinus or set of cells is affected by the chronic catarrhal inflammation and another by the suppurative variety. In such cases I believe the latter is the secondary event, but whether merely in point of time or because the catarrhal inflammation makes the membrane liable to suppurative inflammation, I am not prepared, as yet, to say.

The same observations which lead me to conclude that polypi only arise in cases of catarrhal inflammation, also lead to the conclusion that the two varieties of inflammation are separate and distinct; but there are other evidences which point the same way. We have to consider the change made from chronic suppurative to chronic catarrhal inflammation and vice versa, and the change from acute inflammation to chronic and its opposite.

A case of typical suppurative sinusitis, either acute or chronic, has never, under my observation, changed into a catarrhal sinusitis with formation of polypi. The negative is always difficult to prove, but until one has observed such a case or read of one sufficiently well described, one is entitled to doubt the positive. On the other hand, there are cases of sinusitis in which pus must have been coming away for years, and yet there is no sign of polypi or of catarrhal inflammation.

Next, on opening an antrum full of pus, and the site of chronic suppurative sinusitis, one rarely finds the mucous membrane polypoid. It is usually very thin. It may be thickened, but, if so, the thickening is general and diffuse and not the irregular swelling seen in early cases of catarrhal inflammation. If suppurative sinusitis were a pre-runner of chronic catarrhal inflammation we should see the transitional stage from time to time on opening the antrum. To consider the opposite—a suppurative sinusitis may be added on to a catarrhal inflammation, and this may happen either in the same sinus or in a different one; also the added suppuration may be acute or chronic. This, however, is not the same as the catarrhal inflammation changing into a suppurative inflammation, nor is the catarrhal infection necessarily a factor in the causation of the suppurative one. It may only be a coincidence, though one of which I should always take serious notice.

Whether there is the same distinction between the acute catarrhal and acute suppurative varieties I do not know. Is the pre-suppurative stage of acute suppurative sinusitis a catarrhal inflammation, or are they distinct from the first?

Certainly, in some cases of acute suppurative sinusitis the early stage is not associated with swelling of and secretion from the nasal mucosa but is characterized by marked dryness of the mucous membrane with intense hyperæmia. We may assume that at this stage the mucosa lining the sinuses is in a similar condition. I have come to look upon these as serious cases and to be glad when a secretion and flow of pus have set in, but I cannot say that there is never an early catarrhal stage in acute suppurative sinusitis. So also I cannot say that an acute catarrhal sinusitis does not change into chronic suppurative sinusitis, but I suspect that neither of these occurs.

If these views of the distinctness of chronic catarrhal and chronic suppurative sinusitis are correct, we are led to consider what are the causative differences between the two. Is it that there are different organisms for the one and for the other? or is it a difference in the reaction of the tissues to the same organisms under varying conditions? To these questions I am not prepared to give answers as yet. In the meantime they bring us back to those with which we started, viz., whether polypi indicate some special form of inflammation of the mucous membrane or are secondary results of some other disorder; and if they are correct they answer these by saying that polypi are not mere symptoms of some other underlying disease but are a part of a special type of inflammation going on within one or more accessory sinuses; to remove them cannot be anything other than palliative treatment. Here theory fits in with experience. To cure the condition we must either act upon the inflamed mucous membrane in order to cause it to involute and the inflammation to resolve, or we must remove the whole of the diseased area. We may try to act upon the inflammation either through the blood-stream, or, locally, by procuring drainage and employing lavage. Till we know more about the pathology of the condition medicinal therapeutics is not likely to take us far. Drainage and lavage may be sufficient for the maxillary sinus; but for the ethmoid it is not practicable. To remove the whole diseased area appears to be our only method at present. Being dissatisfied in many cases with the various intra-nasal operations, and acting on a hint from Dr. Logan Turner, I have operated on three cases from without by a method which perhaps others have also used.

84 Layton: *Nasal Polypi and Inflammation of Accessory Sinuses*

Through the incision described before this Section by Mr. Kisch [6] for drainage of the frontal sinus and after removing, as he does, the nasal process of the superior maxilla, I have removed the lachrymal bone and os planum of the ethmoid with all the ethmoidal cells. The whole time one is lateral to the middle turbinal, which I have been careful not to disturb. In this way one dangerous area, that of the olfactory nerve and the cribriform plate, is undisturbed. Only less dangerous than this is the roof of the ethmoidal labyrinth. The anterior ethmoidal vessels and nerve passing from the orbit into the nose act as a guide which tells me how high one can go. In this way I have removed all the ethmoidal cells back to the body of the sphenoid bone.

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Discussion.—Dr. W. S. SYME said he had taught for years that in all cases of multiple polypi in the nose there was antral as well as ethmoidal disease. He opened the antrum in these cases, and had never regretted doing so, for the same changes were there as in the membrane of the nose. He did not agree with Mr. Layton's rigid demarcation of catarrhal from purulent sinus disease, though the catarrhal element might be more evident in one case than in another. Even if there was a negative result from washing out the antrum he was not deterred from opening it. He had so far lacked the courage to do an external operation on the ethmoid, because it might result in some subsequent disfigurement. With careful precautions he thought one could radically operate on the ethmoid from inside the nose.

Mr. C. A. S. RIDOUT said he did not consider that the catarrhal and suppurative types were separate entities, though one case might be more catarrhal, another more suppurative. He agreed with Dr. Syme's remarks about the antrum ; in severe nasal polypi the antrum seldom escaped. He thought the external operation was not done often enough. In one case of his own, after two or three fairly thorough curettings of the ethmoid region, the patient had remained incapacitated. He then carried out a double external operation on the ethmoid, and the patient had now returned to her work.

Dr. J. S. FRASER said that last year he had discussed the recurrence of nasal polypi with Dr. Otto Hirsch, of Vienna. Dr. Hirsch maintained that these recurring polypi came from the maxillary sinus, through an accessory or even through the normal ostium. He (Dr. Fraser), acting on that view, had opened the maxillary antrum in a number of such cases. In several of these the antral mucosa had been polypoid, and polypi could be seen passing into the middle meatus through the accessory ostium. But in one or two of the cases the mucosa of the maxillary sinus was healthy and certainly not polypoid. He therefore felt doubtful about performing the Caldwell-Luc operation in all cases of recurring nasal polypi.

Many years ago he (Dr. Fraser) had published a paper on chronic inflammatory œdema of the submucous tissues of the nose,[1] i.e., polypoid middle turbinals and so-called "hypertrophy" of the inferior turbinal which did not subside on applying cocaine and adrenalin.[1] The inflammatory change was really the same in the two conditions. It was a chronic inflammatory œdema, spreading in the deeper layers of the submucosa, between the superficial layers of epithelium on the one hand, and the glands and blood spaces on the other.

He agreed with Dr. Syme that there was no essential difference between chronic catarrh and chronic suppuration. In the suppurative form leucocytes were present in the discharge in large numbers, while in the catarrhal they were comparatively few. It was a question of the virulence of the infection and of the resistance of the patient—not a real difference in the pathological process.

As to radical operation in these cases, radiograms could not always be depended on: a better criterion was furnished by proof-puncturing the antrum and washing it out. If the

[1] *Journ. Laryngol., Rhinol. and Otol.*, 1908, xxiii, 402.

infected fluid returned easily, the antrum was not filled with polypi; but if one had to exert pressure with the Higginson's syringe the mucosa was probably polypoid and the radical operation was required.

Mr. JAMES ADAM said that not enough emphasis was laid upon the effect of pressure in causing polypi. If the uvula was caught between the two enlarged tonsils, it became polypoid. If there were inflammatory swellings about the middle turbinal, and the mucosa was compressed between the septum and outer wall, polypi formed, and they continued to form until the pressure was relieved by dealing with the septum, or the outer wall, or both.

Mr. F. B. GILHESPY said he was interested in hearing that it was possible to perform an external operation in such a case through a small incision. He had always used a longer incision than Mr. Kisch did, and turned the eye outwards, so as to get back to the sphenoid. Patients required a guarantee that there would not be a recurrence of polypi, therefore the operation must be a thorough one. He would like to hear statistics relating to the Sluyder operation, which would give an indication of its dangers.

Professor PORTMANN said that in most cases polypi of the nose had a connexion with the ethmoid, especially when they were multiple. Sometimes when the nasal polypus had been removed it was necessary to operate on the antrum. In diagnosing maxillary sinusitis one could begin by puncturing and washing out the sinus, and with the sinuscope one could see the condition of affairs. Removal of ethmoidal cells could be carried out by an intranasa operation. This operation did not destroy the sense of smell, and was not dangerous at all when performed with the aid of Moure's curette.

On the Occurrence of Brain-tissue within the Nose: the so-called Nasal Glioma.

By DOUGLAS GUTHRIE, M.D., F.R.C.S. (Edinburgh), and NORMAN DOTT, M.B., F.R.C.S. (Edinburgh).

INTRODUCTION.

THE cranial and nasal cavities lie in such close proximity to each other that one might naturally suppose that both are liable to be involved in the same disease process. The frontal lobes of the brain approach the upper part of the nose very closely in the region of the cribriform plate, and a glance at a frozen section shows that this structure lies at a lower level than might be suspected.

The cribriform plate is subject to considerable variations. As viewed from the upper aspect, it may be broad and practically continuous with the orbital plates. More frequently, however, it lies at the bottom of a deep groove, the sulcus olfactorius, and it may be almost hidden from view, so that the olfactory bulb lies in a sort of tunnel rather than in a groove or depression.

Under the name "hypertelorism," D. M. Greig [1] has described a condition in which the nasal bridge is enormously wide and the ethmoidal labyrinths are absent, while the cribriform plate is extremely broad and projects downwards to the nasal cavity.

Although the foramina of the cribriform plate vary greatly in number and size, congenital dehiscences or defects in this partition are rare, and so far as we can ascertain, the only recorded case of hernia of normal brain-tissue through such an aperture, is that mentioned by Cruveilhier [2]. He discovered, at a post-mortem examination, a hernia of the dura mater and brain which entered the nasal cavity through the cribriform plate, and resembled a nasal polypus. Perhaps at the present day this would be classed as an encephalocele. In rare instances fracture of the anterior fossa of the skull may favour the occurrence of a hernia at the site of the fracture, and only last month a case of this nature was reported to the Section of Laryngology by Mr. Mollison [3].

86 Guthrie and Dott: *Brain-tissue within the Nose*

The upward extension, towards the brain, of a nasal malignant growth, has been recorded. White and Brunton [4], for example, recently described a carcinoma which arose in the olfactory mucosa, in a woman aged 53, and which extended so far as to involve the frontal lobes of the brain.

On the other hand, the downward extension of a brain tumour towards the nasal cavity, appears to be a very rare event. The writers have sought in vain for any record of such a condition and have been unable to discover in museums any specimen similar to that described in the present paper. (Case I.)

A swelling containing brain-tissue, congenital in origin, may occasionally be seen in the frontal region of infants, or still more rarely, may be found within the nose. Such "tumours" are classed as frontal or intranasal encephalocele, although they cannot be regarded as herniæ, as the tissue is not originally intracranial, but is extruded before the skull is developed. Such encephaloceles are caused by faulty development during embryonic life, so that a small bud of brain-tissue grows out from the primitive brain. During the subsequent development of the skull, this "bud" may become separated entirely from the main mass, or may retain a connexion, through a stalk or pedicle, with the central nervous system.

The "polypus" which we removed from an adult patient (Case II, described below), and which was found on examination to contain glial cells, probably falls into this category, although the majority of similar cases previously reported, have occurred in infants, and have been recognized shortly after birth.

These congenital tumours can hardly be regarded as gliomata, as the cells found within them are fully developed glial cells which show no embryonic characters such as would suggest the diagnosis of a neoplasm. Nevertheless, the majority of these cases are reported in literature as "glioma of the nose," "intranasal glioma," etc., although they are different in microscopic structure from the true cerebral gliomata, of which Case I is an example.

Before making further reference to the literature of this subject it may be convenient to give some details of the cases which have come under our personal notice.

Case I. Glioma of the Frontal Lobe of the Brain, with Extension into the Left Nasal Cavity.

History.—F. M., male, aged 33, came under the care of one of us (N. M. D.) on account of the sudden onset of coma with symptoms suggestive of cerebral compression. We are indebted to Professor Edwin Bramwell for notes of the previous history. No previous illness of importance. No history of nasal or aural disease. Deafness in left ear immediately following gun explosion. Two months ago slight generalized headache with nausea—continued—varying in intensity, but rarely absent. Localized chiefly behind eyes, in left temporal and in sub-occipital regions. Three weeks ago headache became more intense and was accompanied by vomiting on several occasions. One week ago, examination by Professor Bramwell showed some drowsiness, retardation of cerebration, pulse 54 to 60, temperature consistently subnormal, very slight early evidence of bilateral choked disc, definite weakness of right arm, slight dysmetria of right arm, slight increase of right knee-jerk; no aphasia, no visual field defect, no weakness of face. X-ray showed no definite abnormality. Twenty-four hours ago, marked increase in drowsiness.

Examination.—September 10, 1925. Personal observation—a well-built male, aged 33, semi-comatose. Slight distension of scalp veins and œdema of scalp about vertex. Pulse 45 to 60 yesterday, has increased to 90 to 100 to-day; respirations deep and sighing; temperature has risen to 100° F. to-day. Bladder distended, retention of urine, catheterized. Definite choked disc in left eye, changes present but less marked in right. Right knee and ankle jerks slightly increased. No facial weakness. No obvious asymmetry of muscular tone.

Diagnosis.—From nature and brevity of history, rapidly growing glioma—in which hæmorrhagic degeneration occurred a few days ago,—of left cerebral hemisphere, probably

frontal, from absence in history of definite visual, speech, sensory or motor changes. Immediate operative relief of intracranial pressure was decided upon.

Operation.—September 10, 1925. Left subtemporal decompression (Cushing). The tense brain was difficult to control during opening of the dura and subsequent closure of the muscular layer. On exposure the cortex was typically pale and œdematous, with dry surface and flattened convolutions. Root of Sylvian fissure displaced upwards by enlargement of inferior frontal region. The exploring cannula was passed to the sites of the temporal and frontal horns of the lateral ventricle but no fluid was obtained, indicating compression of the ventricle by an adjacent tumour. A puncture in the inferior frontal convolution met resistance at a depth of 3·5 cm., and the withdrawal of the cannula was followed by slight oozing of blood, suggesting a deep-lying vascular tumour. As the neoplasm was almost certainly gliomatous, no attempt was made to expose it. The wound was carefully closed over the tense brain. The operation was well tolerated. The fact that the temperature had continued to rise during the operation, reaching 102° F. at its termination, was regarded by us as a significant and ominous feature.

Post-operative Course.—This was satisfactory for the first few hours, during which the temperature fell to 100° F. Fifteen hours after operation the temperature rose abruptly to 105·4° F., accompanied by rapid feeble pulse and distressed breathing. The temperature was immediately reduced by cold sponging and exposure. Its fall was accompanied by marked improvement in the general condition. On the following day a similar hyperthermic attack occurred and was similarly treated with satisfactory result. During the ensuing week the patient had to be constantly exposed at the open window and sponged at frequent intervals; any remission in this treatment permitted the temperature to rise to a dangerous level. With artificial regulation of the body temperature the patient's condition remained satisfactory. Some degree of consciousness was regained. He could understand what was said to him, could appreciate facial expression, and could speak sufficiently to make himself understood. He expressed himself as entirely comfortable. The wound healed soundly. The decompressed area protruded progressively with extraordinary rapidity. On the eleventh day right facial weakness, temperature rising, pulse weak and irregular, breathing of "Cheyne-Stokes" type. On the twelfth day condition worse, complete right hemiplegia, temperature 103° F. in spite of treatment, consciousness again lost. On the thirteenth day he died, the temperature having reached 105° F.

The clinical course after operative relief of tension was obviously due to a very rapidly growing tumour which was invading the hypothalamic region of the base of the brain (control of temperature, heat production by endogenous metabolism and surface loss by sweating) giving rise to the typical cerebrogenic hyperthermia. Finally, the tumour growing backwards had invaded the motor fibres of the internal capsule.

There had been no complaint of nasal disturbance in the history; the olfactory function had not been investigated preceding the onset of coma. When the patient came under our observation such investigation was precluded. It would certainly have been of great localizing value in this case as the description of the post-mortem findings will show.

Post-mortem Examination: Twelve hours after death. After the vertebral arteries had been clamped, 2 litres of 10 per cent. formol solution were run into the common carotid arteries in the cephalic direction. The skull cap was then removed. The area of the operation was in excellent condition. The surface of the brain showed marked convolutional flattening and a sharp mushroom-like projection at the decompression site. Upon the dura along the longitudinal sinus being separated from the brain, and later upon elevation of the temporal lobes from the dura of the base, numerous minute cerebral herniæ were encountered, constituting points of adhesion which broke away readily, usually through the delicate neck of the hernia, which remained in its minute dural pocket. On elevation of the frontal lobes, the left lobe was found to be adherent to the base of the skull in the region of the left olfactory groove. The adherent area could not be detached without injury to the brain. It was therefore cut through, being left attached to the skull, and the removal of the brain completed in the usual

88 Guthrie and Dott: *Brain-tissue within the Nose*

FIG. 1 (*a*).

FIG. 1 (*b*).

manner. The sella turcica showed slight flattening and expansion. The ethmoidal region of the base of the skull was removed with the chisel. The brain was further fixed by suspension for ten days in 10 per cent. formol solution and was then cut in coronal sections.

Pathological Appearances.—Fig. 1 (*a*) shows the appearance of the base of the brain.

Note the marked displacement of the median structures of the base to the right side, and the venous engorgement of the left hemisphere as contrasted with the right. This is possibly due to compression of the superficial middle cerebral vein by the tumour which immediately underlies its lower end. Note in the region of the left olfactory lobe the laceration of the brain surface. This is where the adhesion in the region of the left olfactory groove has been separated. In the hypothalamic region just in front of the optic chiasm there is marked swelling due to the projection of the tumour upon the surface. The entire left hemisphere

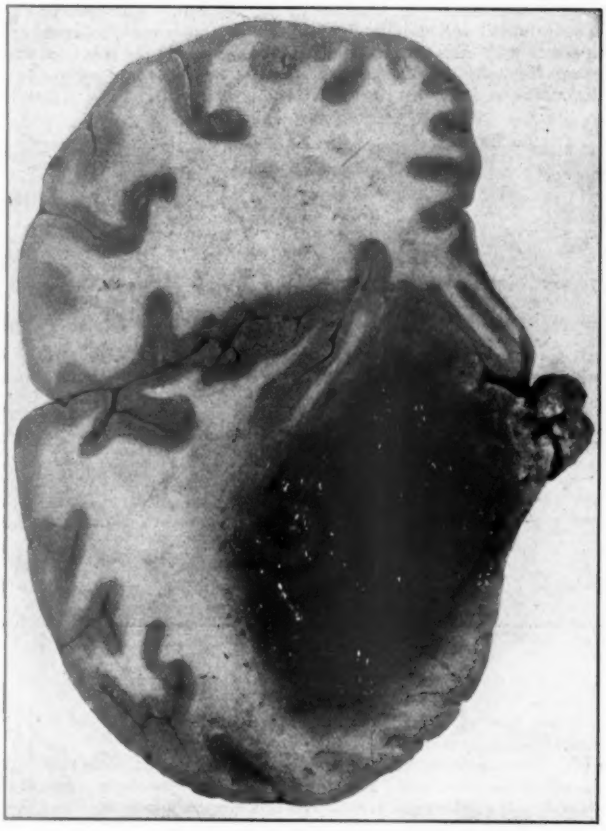

FIG. 2.

shows marked swelling and its projection into the decompression is seen. (*b*) Shows the intracranial surface of the ethmoidal region of the skull, with dural lining. Note the deviation of the anterior attachment of the falx to the right side. The left olfactory groove is replaced by a large excavation in which tumour tissue is seen adherent to the expanded dura.

Fig. 2.—Coronal section through frontal lobes.—P. A. view. Note the large infiltrating glioma with marked degenerative changes and extensive hæmorrhage into its substance.

90 Guthrie and Dott: *Brain-tissue within the Nose*

It has produced a gross asymmetry of the brain by enlargement of the left inferior frontal region. Note especially the marked projection of the tumour in the left ethmoidal region, which constituted a hernia into the left nasal cavity.

Coronal sections further back show involvement of the hypothalamic region, left corpus striatum and anterior part of internal capsule. The third ventricle has been compressed laterally, and, as a consequence, a considerable distension of the lateral ventricles is present, with the exception of the frontal and temporal horns on the left side which had been directly compressed by the adjacent tumour.

Microscopic Appearances.—Fig. 3 shows the microscopic characters of the growth. The neoplastic cells are small and spindle shaped; the protoplasm is moderate in amount; there is an early indication of fibril formation at their poles; nuclei are oval and stain deeply; mitotic figures are frequent. The cells exhibit considerable irregularity of size and development, and degenerative changes are everywhere apparent. The growth possesses no

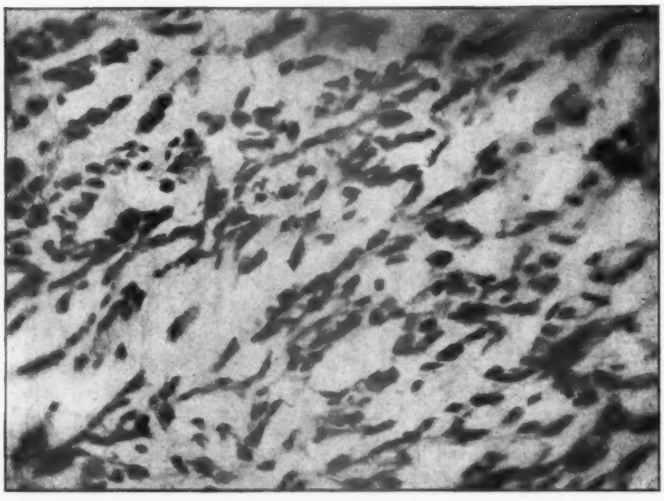

FIG. 3. × 340.

recognizable stroma nor architecture; many parts show evidence of intercellular œdema. The blood-vessels are extremely primitive.

The margins show characteristic invasion of the adjacent cerebral substance, which presents the usual gliosis reaction. The tumour cells can be seen penetrating between the layers of the dura at the adherent area in the left olfactory groove. The periosteal layer of the dura appears to be intact. The picture is that of a rapidly growing malignant neoplasm; the cell type involved represents the early embryonic spongioblastic phase in the development of neuroglial tissue. It is a spongioblastoma. The clinical malignancy of the growth and its primitive cytologic type are in accord.

Fig. 3 is of especial interest from the rhinological standpoint. It represents a section through the gliomatous protrusion which had penetrated the left cribriform plate and entered the nasal cavity. It shows advanced degenerative changes in the tumour cells. This degenerative tissue presents very active infiltration by polymorphonuclear leucocytes, an unmistakable evidence of early pyogenic infection—probably directly from the nasal cavity. The process is extremely early and is limited to an area little larger than the microscopic field shown. It represents the earliest stage in the formation of a cerebral abscess. It

played no part whatever in the clinical course of the case, but it is of interest to reflect that, had the patient survived longer, he would almost certainly have developed a cerebral—or rather a "gliomatous" abscess as a consequence of invasion of the nasal cavity by a tumour of the brain.

Commentary.—This case represents what we believe to be a rare cause of the presence of gliomatous tissue in the nasal cavity—the direct penetration of a cerebral glioma through the cribriform plate. It is generally recognized as characteristic of glioma that it does not invade (in the pathological sense) tissues other than those of the central nervous system—hence it does not metastasize as do other malignant growths. The dura mater usually forms an impassable barrier to the glioma. The penetration of the tumour to the nasal cavity in this instance, therefore, requires explanation.

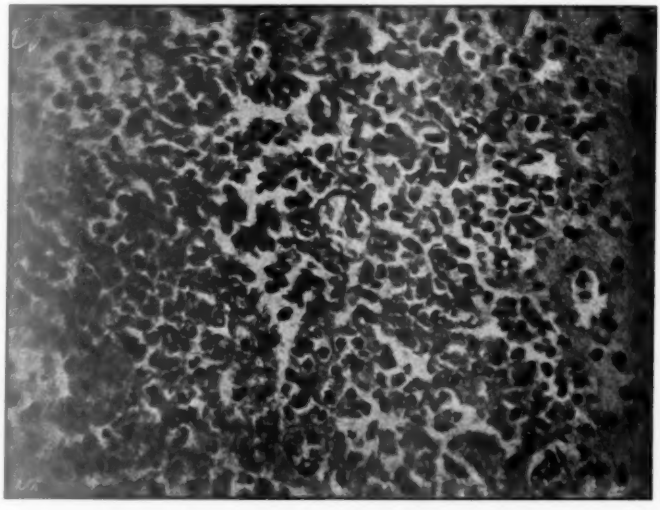

FIG. 4. × 200.

In cases of long-standing intracranial hypertension the occurrence of multiple cerebral herniæ is not uncommon. In such cases the herniæ consist of small polypoid masses of cortical substance which have gradually penetrated the minute natural apertures of the inner layer of the dura in the walls of the venous sinuses for the passage of the arachnoidal villi. They are observed most frequently along the sagittal sinus and its lateral lacunæ, and at the inner part of the temporal poles over the cavernous sinuses. We have observed them exceptionally attaining dimensions of about 3 mm. in diameter. They are not confined to the locality of a tumour, but are symmetrically distributed in the above-mentioned regions. They are to be found in cases of chronic hydrocephalus in the absence of tumour. They are formed under the influence of the pressure of the soft cortical substance against the dura, distending its minute natural apertures. Villi are numerous about the crista galli, and the hernial protrusions are common here. We may reasonably suppose that the nasal protrusion began in this way—probably before the glioma had reached the

92 Guthrie and Dott: *Brain-tissue within the Nose*

surface of the brain. Having reached the surface by invasion of the brain substance, the neoplasm would certainly involve the cortical hernia, and thus gain a footing *in* the dura. With the characteristic proliferative activity of a neoplasm it would expand and weaken the membrane. The bone—much less resistant to such pressure than the dura—would naturally undergo pressure erosion, and thus the growth would penetrate the nasal cavity. One of us (N. M. D.) has observed an entirely analogous penetration of the superior longitudinal sinus by a glioma.

Case II.—*Nasal Polypus which was found on Microscopic Examination to Contain Brain Tissue.*

History.—A brief record of events prior to removal of the polypus will be sufficient.

J. S., a well-built man, aged 46, previously healthy, was struck by a falling beam of wood in August, 1911. He was rendered unconscious for a short time and the nose bled profusely, but he rapidly recovered from the concussion.

During the following year, 1912, there gradually appeared paralysis of the right arm and leg. The first symptoms were a tingling sensation and a feeling of weakness in the right arm, which gradually became worse. Three months later, there was a similar affection of the right leg, and walking became difficult.

In the following year, the hemiplegia became worse and since then it has persisted. At the onset of the illness, there was slight paresis of the face, but this soon passed off. There was frequent complaint of headache and occasionally, at the onset, there were attacks of vomiting.

The discs were repeatedly examined but there was never any sign of optic neuritis. There was no hemianopia, no giddiness and no nystagmus. The patient's speech was slow and hesitating, and his mental outlook altered so that he became childish and emotional. Wassermann test of blood and cerebro-spinal fluid was negative, and the prolonged administration of large doses of potassium iodide produced no effect.

At this stage, the condition was regarded as glioma of the left frontal lobe of the brain, a diagnosis which was negatived by the subsequent history.

In July, 1920, the patient was found lying unconscious in the bathroom. He remained unconscious for five days and then gradually recovered, the paralysis remaining unaltered.

It was only towards the end of 1920 that he began to suffer from nasal obstruction on the left side.

In November, 1920, and again in July, 1921, several polypi were removed under local anæsthesia, from the left nasal cavity. On naked-eye examination they appeared to be the common type of nasal polypus, and the tissue was not examined microscopically.

Nasal Condition.—Early in 1925, the signs of left-sided nasal obstruction reappeared, and at this stage the patient first came under the care of one of us (D. G.) A soft, greyish tumour, with several small vessels on its surface, occupied the upper part of the nasal cavity. The lower edge of the tumour reached almost to the floor of the nose and only a small portion of the inferior turbinal was visible. There was no pus in the nose or nasopharynx. On posterior rhinoscopy the polypus could not be seen and the nasopharyngeal structures were normal. There was no alteration of the external nose. Apparently only one polypus was present, and the site of origin was probably the middle turbinal. No radiogram was taken. The condition was regarded as a large nasal polypus of the common type.

Operation.—On October 23, 1925, chloroform was administered and the polypus, together with a small portion of middle turbinal, was removed in one piece, with Luc's forceps. It was rather larger than a pigeon's egg, but during removal it burst, and discharged about a drachm of straw-coloured fluid. Hæmorrhage was comparatively slight and soon ceased. The pedicle of the growth was small but the exact site of origin could not be determined.

Subsequent History.—Following operation, the nasal obstruction was entirely relieved and the patient stated that he felt much better. He was seen again a month later, when there was some scar tissue at a point corresponding to the root of the middle turbinal, but otherwise the nose appeared normal.

Early in 1927, that is, sixteen years after the onset of the hemiplegia, and seven years after the first appearance of the nasal polypus, the patient was reported to be in fair physical

condition. He was able to walk about a little and even to work in his garden. The hemiplegia had remained unaltered for several years and the mental condition was in no way improved. There was no complaint of headache or of nasal obstruction.

Pathological Report.—The unusual nature of the growth was not suspected before operation. At the operation, however, the tissue seemed to be more solid and fleshy than is

FIG. 5.

FIG. 6. × 10.

usual in a nasal polypus, and it was therefore retained for examination. After fixation, as fig. 5 illustrates, it was the size of a walnut, rounded, with smooth surface and presenting a ragged and fibrillary appearance at the point of attachment.

The accompanying photographs (figs. 6-10), kindly prepared by Dr. James Dawson, illustrate the microscopic structure of the polypus.

94 Guthrie and Dott: *Brain-tissue within the Nose*

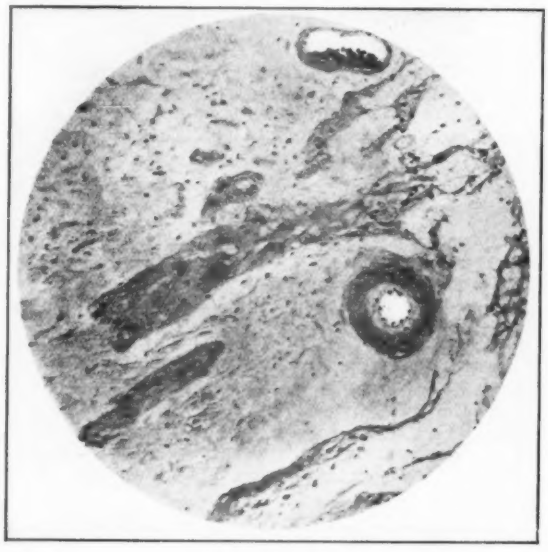

FIG. 7. × 100.

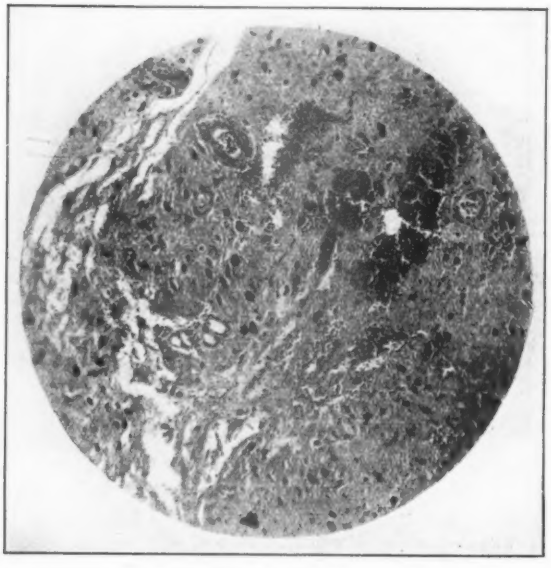

FIG. 8. × 100.

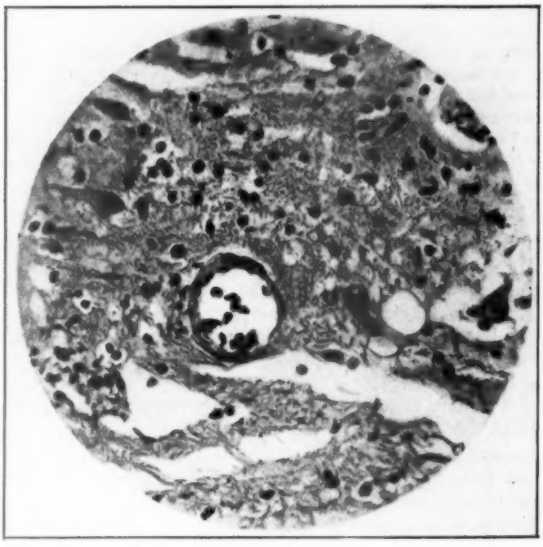

FIG. 9. × 450.

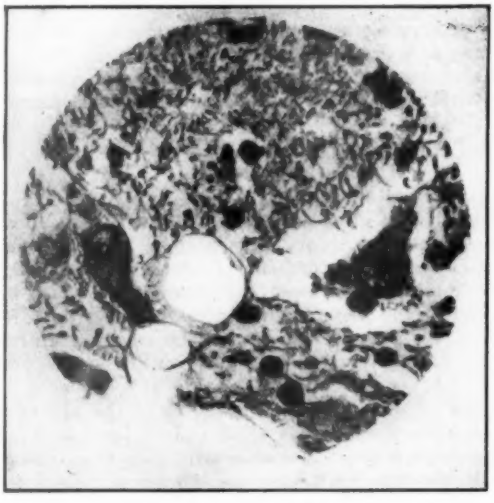

FIG. 10. × 700.

96 Guthrie and Dott: *Brain-tissue within the Nose*

In a section passing through the entire mass, three layers may be distinguished (fig. 6). The surface is clothed with ciliated epithelium while the submucosa is œdematous and infiltrated with small branching connective tissue cells. Beneath the submucosa is a layer of fibres, and of cells, the staining reactions of which suggest hyperplastic meninges; the fibres predominate. In the central portion the fibres gradually give place to cells, and in this region may be seen the glial cells which characterize the tumour. Fig. 7 shows several strands of glial tissue invading the connective tissue of the submucosa.

A high power view (fig. 8), shows four or five large glial cells of typical appearance. The majority of the cells have lost their surrounding protoplasm and appear merely as nuclei. Under oil immersion (fig. 9) the structure of the glial cells may be still more clearly seen.

Commentary.—When the true nature of the polypus removed from this case was first discovered, it was thought that it might bear some relationship to the glioma of the frontal lobe of the brain, which was diagnosed in the early stages of the patient's history. This diagnosis, however, was negatived by the long duration of the symptoms, the patient having remained for years in the same condition without any obvious increase of intracranial pressure. It is more probable that the tumour within the nose had nothing to do with the intracranial lesion. Whether that lesion was due to a slowly-growing meningioma or to a traumatic cyst of the brain, is uncertain. Both conditions are compatible with the symptoms observed, and with the long duration. For the purposes of the present paper, the nasal growth is of greater interest than the brain lesion. It is certainly composed of glial tissue, but the cells are fully formed and show no embryonic characters such as would suggest a true neoplasm or glioma.

As may be gathered from a study of the literature (see below), the term "glioma of the nose" is a misnomer. The existence of glial tissue in the tumour is undoubted, and it corresponds in microscopic appearance to those cases described by Anglade as "fibrogliome," but it would be more correct to retain the term "encephalocele." The glial tissue may have been present within the nose since birth, and may thus have become incorporated in the nasal polypus. It is difficult to explain its existence on other grounds, and in searching the literature on the subject we have failed to discover any similar condition in adult life, although many analogous cases in infancy are reported. It is not improbable that the systematic investigation of all nasal polypi would reveal the presence of nervous tissue in a certain proportion.

Records of Similar Cases in Literature.

The case which we have recorded as Case I was a glioma of the brain, with intranasal extension, and as already mentioned, we could find no record of any glioma which showed this peculiar tendency.

As regards Case II a number of intranasal tumours containing nervous elements have been described from time to time. The congenital swelling known as encephalocele, not uncommon in the occipital region, may appear on rare occasions in the fronto-nasal region, and still more rarely may be entirely intranasal. As already noted, those swellings are neither herniæ of the brain nor true gliomata. They are formed during early embryonic life, before the primitive elements of the skull appear, by the outward growth of a small bud of tissue from the anterior cerebral vesicle. When at a later stage the membranous skull is laid down, this bud of tissue may prevent the complete separation of the ectoderm from the neuro-epithelium, and a foramen may remain, through which the encephalocele retains its connexion with the brain. In other cases, this connexion is not retained, and the bud of nervous tissue becomes entirely cut off from the parent structure.

Paul Berger [5], in whose paper will be found a complete account of the pathology of encephalocele, recognized that certain encephaloceles were not merely miniature brains surrounded by meninges. He stated that sometimes the meningeal envelope and the nerve cells were so altered that it was justifiable to regard the tumour as a neoplasm, for which he proposed the term "encephaloma." He was of opinion that there existed a series of transitional forms, between the encephalocele and the encephaloma, and that, in consequence, the clinical classification was often a matter of difficulty.

There has, however, been some confusion in the writings of subsequent observers regarding the nomenclature of nasal encephaloceles and a number of cases have been described as nasal gliomata.

M. B. Schmidt [6], for example, reports a "glioma" the size of a hazel nut, in the fronto-nasal region of an infant aged 10 weeks. It was easily excised, and presented no connexion with the brain. Schmidt quotes E. Meyer [7] who described a "congenital ethmoidal encephalocele" which consisted of a polypoid intranasal projection of glial substance, and also Reid [8], who observed a similar case in which connexion with the brain was maintained by a stalk which traversed an aperture in the position of the foramen cæcum.

Payson Clark [9] described two congenital tumours which had the structure and staining reactions of gliomatous tissue (? glial tissue). The first was a small rounded tumour of the base of the nose in a boy aged 2, whose left nasal cavity was obstructed by a pinkish, polypoid growth. In the second case, the patient, a male child aged 10 weeks, had suffered from nasal obstruction since birth. A pink swelling projected from the left nostril.

Sussenguth [10] regarded as "glioma," a swelling which he excised from the base of the nose of an infant aged 11 days. It contained glial cells and was unconnected with the brain.

Dahmann and Müller [11], in their interesting study of injuries to the cribriform plate, mention the case of a child aged 2, who died of meningitis four weeks after snare-removal of a nasal polypus. At post-mortem examination there was found a dehiscence in the cribriform plate through which passed the stalk of an intranasal encephalocele, which had been mistaken for a polypus and partly removed.

Perhaps the most noteworthy contribution to the subject under review was made by Rocher and Anglade [12] who described five cases of "fibroglioma of the nose" which they had observed personally. In three cases the tumour was extranasal and appeared at the root of the nose; in one case it was entirely intranasal, and in the remaining case the growth appeared in both situations. The intranasal fibroglioma was previously recorded by Anglade and Philip [13]. It occurred in an infant three days old, the right nostril being obstructed by a small, smooth, red tumour, which projected from the orifice. The left nostril was blocked by deflections of the septum caused by pressure of the tumour. The growth was successfully removed with a snare. The swelling in all cases was congenital, and was recognized in early infancy. Operation was successful in all, and in two of the extranasal cases it was necessary to ligature a small pedicle from which cerebro-spinal fluid flowed freely. Histologically, the tissue consisted of neuroglia, fibrous tissue and blood-vessels. Anglade regards these congenital swellings as composed of cerebral tissue which has come to lie outside the skull during development. They constitute a variety of encephalocele for which he proposes the term "fibroglioma," an unfortunate term, as their structure in no way resembles neoplasm and they only occasionally undergo any increase in size.

It will be noted that each of these recorded cases was congenital and was recognized in infancy. Consequently it is difficult to determine the relation of such tumours to the tumour described in the present paper as Case II. We have only found one record of an intranasal tumour of nerve origin, in the nasal cavity of a patient over three years of age. This appears in a paper by Terplan and Radofsky [14]. The tumour, which was a neuro-fibroma, was removed, by the antral route of approach, from the left ethmoidal region of a girl, aged 13, who had suffered from nasal obstruction for seven weeks. In histological appearance it was similar to the well-recognized tumour of the nervus acusticus.

98 Guthrie and Dott: *Brain-tissue within the Nose*

SUMMARY.

(1) The occurrence of brain tissue in the nasal cavity as a development from an embryonic rest of the central nervous organ, is sufficiently common in young children to constitute a definite clinical entity.

(2) Apart from such cases of encephalocele, intranasal tumours containing glial tissue are extremely rare.

(3) Case II was probably one of a small encephalocele which in adult life became incorporated with nasal polypus. It could not be regarded as a glioma, as the cells showed no neoplastic characters. No previous record of such a case had come to our notice.

(4) Case I, on the other hand, was that of a nasal glioma. It illustrates the penetration of the nasal cavity by a cerebral frontal glioma of the spongioblastic type. The mode of penetration has been explained on the basis of neoplastic invasion of a pre-existing minute pressure hernia of the cortex. The occurrence of early pyogenic infection of this gliomatous protrusion has been demonstrated.

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Discussion.—Dr. LOGAN TURNER said he had removed the polypi in the second case some years previously. They had the appearance of ordinary nasal polypi. Was it not possible that in this case there might be a large congenital gap in the cribriform plate, and that there might originally have been an encephalocele projecting through it?

Professor GEORGES PORTMANN (Bordeaux) said that such cases were seldom seen in Bordeaux, but one or two had been observed, the patients being infants. Mr. Guthrie's were particularly interesting because the patients were adults. The question was whether the condition was one of simple glioma or of glio-sarcoma. In the present case there seemed to be evidence of malignancy.

Dr. J. S. FRASER asked whether in such cases there was anything in the appearance in the nose which would warn the observer that he was dealing with something beyond ordinary nasal polypi.

Mr. DOUGLAS GUTHRIE (in reply) said that the paper described two different conditions. The first was a glioma of the frontal lobe, which invaded the nose. The second he did not regard as glioma, but as an encephalocele which developed in the nose, probably in embryonic life, and later became involved in a nasal polypus. To find such a condition in an adult was very unusual. Possibly the patient had "ordinary" nasal polypi previously. Clinically, the polypus could not be distinguished from the common type, though it was rather tough and fibrous. The microscope alone gave a clue to the unusual nature of the tissue.

Section of Laryngology 99

Meningitis of Sphenoidal Sinus Origin.

By M. VLASTO, F.R.C.S.

IN considering this subject, I am excluding those cases of meningitis which are the result of meningococcus infection and which give rise to the condition known as epidemic cerebro-spinal fever.

In 1920 Embleton reported that out of thirty-four necropsies of persons dying from cerebro-spinal fever, empyema of the sphenoidal sinuses was found in thirty. And Peters reported seven cases of cerebro-spinal meningitis in which in five cases there was recovery after the sphenoidal sinus had been opened and drained. Whereas in searching through the literature on the subject, I have been unable to find a single case in which a meningitis of non-meningococcic origin has been attended with recovery, with or without operation.

It seems reasonable to assume that in the first case the sphenoidal sinus can be regarded as the carrying site of organisms of a non-thrombosing type and frequently of a low degree of virulence, whereas in the case of meningitis due to infection by other pyogenic organisms the sphenoidal sinus is an active seat of inflammation due to organisms of which the virulence is very high.

It is this latter variety which is considered in this paper.

Apart from certain rare terminal sequelæ, the intracranial complications of sphenoidal sinus suppuration fall into one of the following three groups.

(1) Meningitis; (2) thrombo-phlebitis of the venous sinuses, cavernous, longitudinal or both; (3) meningitis and thrombo-phlebitis.

The first important contribution in this country to the investigation of the sphenoidal sinus origin of an intracranial infection was made by StClair Thomson, in a valuable paper published in the *Transactions of the Medical Society of London*, in 1906, in which he collected records of forty-two cases of sphenoidal sinus infection ending fatally from intracranial infection. Of these forty-two cases, at least thirty patients died of basal meningitis, and in seventeen cases this was the only or predominant lesion.

An important contribution to this subject, abroad, made by Toubert in 1900, preceded StClair Thomson's paper. Toubert collected records of twenty-four cases of intracranial complications of sphenoidal sinus origin. Some of these cases are naturally referred to in StClair Thomson's communication. Of these twenty-four cases, nine showed basal meningitis, or at any rate this was the predominant lesion.

In 1909 Roughton briefly reviewed the subject in the *Clinical Journal*. In addition he quoted a case of his in many respects identical with a case of my own which I will now describe.

Patient, a girl aged 11, was admitted to the Queen's Hospital for Children on October 29, 1926.

History.—The child had been ailing for three weeks previous to admission. Her feverish complaint had been diagnosed as influenza. She had complained of headache and pain in the left ear, which discharged for five days and then had cleared up.

Past History.—She contracted scarlet fever three years ago, but the senior assistant medical officer of the North-Eastern Fever Hospital assured us that the case was a perfectly straightforward one of mild scarlet fever without complications. Nevertheless, ever since suffering from this fever, so the history stated, the child had had a non-fœtid discharge from both nostrils and was constantly blowing her nose. She attended the out-patient department of the Central London Hospital for this complaint in 1924, but beyond slight crusting inside the nasal fossæ, nothing abnormal was found. (I think I should add that this history of nasal discharge was only obtained posthumously.) Lastly (a point which may, or may not, have its importance), the father stated that for the last two years the child's scalp was so sensitive that combing the hair caused acute pain.

100 Vlasto: *Meningitis of Sphenoidal Sinus Origin*

Condition on Admission.—Temperature 104·6° F. She was complaining of severe headache referred to the frontal region. She was fully conscious but cerebration was slow. The neck was slightly stiff and the child resented interference. There was nothing particular to note in the central nervous system. The clinical picture was that of early meningitis.

Aural Condition.—The appearance of the right drum was normal. The left drum was injected, but no discharge was present and no perforation was seen. There was no evidence of mastoid involvement.

The nasal fossæ were not inspected (particular attention is drawn to this omission).

Diagnosis at this stage lay between meningitis of otitic origin or a tuberculous meningitis.

In view of the definite history of a recent left aural discharge, and the appearance of the left drum, it was decided to perform a left radical mastoid operation and be guided by the findings.

A lumbar puncture was carried out before the operation on the day of admission. Several c.c. of turbid fluid under pressure were withdrawn, and 4 c.c. of horse's serum were injected into the thecal canal.

The radical mastoid operation showed the tympanum, antrum and mastoid cells to be free from macroscopic disease. The dura mater over the tegmen antri was freely exposed, and as it appeared normal it was not incised.

The pathological report of the cerebro-spinal fluid was as follows: *Cells:* a large number of polymorphonuclears. *Sugar:* absent. *Chlorides:* 0·64 per cent. *Culture:* streptococci.

The diagnosis to be made was now clearly one of septic meningitis, and I had a very natural feeling that I had not got to the bottom of the trouble.

Subsequent Course of the Illness.—The patient went steadily downhill. On November 1, that is to say, three days after admission, 100 c.cm. of blood were injected into the median basilic vein from a Group IV donor kindly provided by the Red Cross. I was influenced in having this transfusion carried out by reading in the *Archives Internationales* an article by Maduro reporting the cure of three cases of otitic septicæmia by the transfusion of a relatively small quantity of blood.

Treatment, however, had no effect on the disease, and the patient died two days later, that is to say, five days after admission.

Post-mortem Findings.—A condition of basal meningitis was present, a thick layer of pus spreading between the region of the optic chiasma and the pons. Section of the brain was negative, and the intracranial adnexa of the ears were normal. The venous sinuses were not thrombosed.

So little did the possibility of sphenoidal sinus infection occur to us, that we were about to regard the meningitis as a pathological expression of a general septicæmia—in other words, as a meningitis of undetermined origin. It was decided, however, to open the sphenoidal sinuses as a routine measure before leaving the post-mortem table.

Both sphenoidal cavities were filled with pus. It is unfortunate that sphenoidal empyema was not suspected, and that no pains were taken to preserve the specimen *en bloc* for further examination.

Failure to diagnose that a meningitis is of sphenoidal sinus origin is not unusual. Indeed, from a perusal of the clinical records of other cases, one can state that the ante-mortem diagnosis is the great exception rather than the rule. There are many reasons to account for this fact.

(1) Patients often overlook the possible gravity of a purulent nasal discharge and fail to report its presence, even when they are conscious enough to do so. This is particularly likely to occur in the case of young children, who are less particular than adults in this respect.

Coakley, commenting on the difficulty in diagnosing a rhinogenetic cause of meningitis, wrote as follows:—

"The rhinologist meets difficulties in his examination of the nose far greater than the otologist in the examination of the ear. Nearly every patient ignores a moderate amount of discharge from the nares, considering it as a type of catarrh common to everyone. Consequently, inflammatory lesions of the accessory sinuses, accompanied by a moderate discharge but unaccompanied by pain, are frequently unheeded by the patient. On the other hand, a discharge from the auditory canal is cause for concern and the seeking of relief."

(2) Another cause for the diagnosis being side-tracked from a pathological sphenoidal sinus condition is the frequent co-existence of a suppurative middle ear. The association between nasal sinus infection and infection of the middle ear has been emphasized by Watson-Williams and others. Also, when Yates investigated the activity of the ciliary epithelium within the accessory sinuses of the nose, he traced, experimentally, the route of infection between the sphenoidal sinus and the middle-ear tract.

Fremel especially emphasizes this point as follows:—

"The subjective and objective symptoms which now make their appearance are referred to the obvious middle-ear disease and its complications, which are in the foreground of interest. It is only when the complications become more serious, and when after the ear has been operated upon, more or less serious disease is found, that the surgeon is left with an uncomfortable feeling that what has been found does not account for the severity of the patient's condition. After the unsatisfying operation the severity of the disease picture becomes greater, and finally the post-mortem examination reveals meningitis originating from the sphenoidal sinus."

Lastly, I submit that even were the sphenoidal sinus suspected as being the focus of infection, the surgeon might hesitate before advising an operation involving the removal of the middle turbinate bone in a patient who was very seriously ill.

Conclusions.

(1) Meningitis of sphenoidal sinus origin may not be so infrequent as supposed.

(2) No post-mortem examination is complete in a case succumbing to meningitis which does not include the opening of the sphenoidal sinus cavities.

(3) In cases in which an otitic cause of the meningitis has been diagnosed, and where the operative findings are inconclusive, the possibility of a sphenoidal sinus infection should be considered.

I am indebted to my house surgeon, Dr. Ivy Ward, for much help in collecting the notes of my case, and to Dr. O'Flynn for carrying out the pathological investigations.

Discussion.—Dr. P. Watson-Williams said that probably there were few who could always diagnose in time the source of infection in these cases. In children, especially, one was apt not to think of the question of sphenoidal sinus infection under such circumstances, and there was a belief that in childhood sphenoidal sinus infections were extremely difficult to deal with. He, himself, explored the sinuses in practically all mastoid cases, and often he found conclusive evidence of previously unsuspected suppuration. Exploration of the sphenoidal sinus did not involve removal of any part of the middle turbinate. It was easy, with the blunt cannula, to enter the sinus and to suck up any contained discharge, first one side and then the other side. In a child, especially, he usually began with the maxillary antrum, and if this was uninfected and there was reason to suspect the sphenoidal sinus, that would be explored, when the presence of infective discharge would be revealed.

As to the route of infection, at first he thought it was through the bony wall, but he now doubted it. Few pathologists appeared to look upon the sinuses as worthy of careful routine examination. In such cases it was important to know the exact route taken by the infection, and therefore the sphenoidal sinuses and ethmoid labyrinth should be removed *en bloc* and submitted to careful investigation. He thought many cases of coccal meningitis were of nasal origin.

Mr. Norman Patterson asked whether the ethmoidal cells were opened and whether they were suppurating.

Dr. Logan Turner thought it very unfortunate that the sphenoidal cavities should be opened at the time the post-mortem examination was made. One could not tell by naked-eye observation the route of infection. Much information could be obtained from the

102 Vlasto: *Meningitis of Sphenoidal Sinus Origin*

examination of the accessory sinuses if the block containing the ethmoid and sphenoidal sinuses were removed, and the diagnosis postponed until a complete microscopical examination could be made.

In this case there did not appear to have been cavernous sinus thrombosis. But many cases of lepto-meningitis associated with sphenoidal sinus suppuration were secondary to septic thrombosis of the cavernous sinus, due to a retrograde septic thrombosis of the veins which threw their blood into the cavernous sinus, many of those veins being meningeal. It was not always necessary that there should be clinical signs of cavernous sinus thrombosis.

What was the direct route of infection from the sphenoidal sinus to the lepto-meninges? One would naturally say by the lymphatics, but so far as he knew no direct lymphatic route had been traced anatomically from the sphenoidal mucous membrane to the pia-arachnoid meshes. It was a point needing further inquiry.

Sir JAMES DUNDAS-GRANT said that in many cases, if the sphenoidal sinus was opened post mortem, a muco-purulent fluid would be found, which was not generated there as a result of inflammation of the sinus, but had entered post mortem when the cadaver was on its back. A little muco-pus in the sphenoidal sinus was therefore not of the same significance as it was when in the ethmoidal cells.

Sir STCLAIR THOMSON said that in a paper, written twenty-two years ago, he had reported two cases with cerebral complications and death—not traumatic or surgical—from sphenoidal sinusitis, which had been diagnosed in life and fully proved by post-mortem examination. He was disappointed to hear that examination of sinuses was not a systematic part of every post-mortem; he hoped those engaged in active practice would persuade their colleagues not to omit this routine. It could be readily done without disfiguring the face of the cadaver. After the skull-cap had been taken off, by sawing down the bone to the root of the nose and behind to the foramen magnum, the two halves of the skull fell away from each other, the sphenoidal block could be removed, and the skull could be put together again and the skin stitched.

He asked whether Mr. Vlasto said he removed the middle turbinal to give drainage? The speaker questioned whether it was wise, in acute sphenoid conditions, to produce any traumatism in the nose beyond a proof-puncture of the antrum, as it had been shown that in acute sinus disease this tended to open the blood- and lymph-channels.

Mr. VLASTO (in reply) said he regretted that, for reasons stated in the paper, no steps had been taken to preserve the specimen of his case *en bloc*. He could not state whether or not the ethmoidal sinuses were infected, because they had not been specifically investigated. The cavernous sinuses were incised, and they had contained liquid blood. He would not care to say definitely that there was not a mural thrombosis. The statement that the meningitis was of sphenoidal sinus origin was based on the facts that the sphenoidal sinuses were filled with pus, that the meningitis was basal in position and extended from the region of the optic chiasma to the pons, and that no other cause for the meningitis was found. He agreed that ante-mortem diagnosis would probably not have saved the patient's life, but this was no excuse for the fact that a diagnosis had not been made. Of the very few cases in which an ante-mortem diagnosis had been made, two of these were reported by Sir StClair Thomson, and in both cases the patients had died. He noted the fact that Dr. Watson-Williams would have proof-punctured the sphenoidal sinuses in a case such as had been reported, but he, personally, had not found sphenoidal sinus puncture in the case of a child either so reliable or so easy to perform as one might be led to believe.

Notes of Three Bronchoscopic Cases.

By W. S. SYME, M.D.

Case I.—Right Bronchus Blocked by a Necrotic and Fibrous Mass. Operation through the Bronchoscope. Recovery. Mrs. E. H., aged 45, was admitted to the Western Infirmary, Glasgow, on July 3, 1925, because of great difficulty in breathing. For two years she had suffered from periodic attacks of suffocation and from what were thought to be asthmatic attacks. During these attacks she became very

cyanosed. There was, too, at times copious expectoration of a purulent nature. She was confined to her bed at first for ten months, but during the past year her condition had improved somewhat, though exertion still brought on severe attacks. The breathing was noisy, suggesting an obstruction in the air passage.

Examination of the chest showed relative dullness on percussion over the right lung behind, with bronchial breathing. X-ray examination revealed extensive fibrosis of the right lung. Bronchoscopy was carried out on July 6. A mass was found blocking the entrance to the right bronchus. From a ragged opening in this mass there protruded necrotic, purulent material. A probe was cautiously passed through this and entered a cavity apparently about the size of a hen's egg. The contents were removed by swabbing, the opening was enlarged by means of punch forceps, and the cavity again mopped out and then swabbed with 10 per cent. silver nitrate solution.

The pathologist reported on the fragments removed:—The fragments consist of necrotic tissue in which there are subacute and chronic inflammatory changes. The exact nature of the lesion is obscure but no evidence of malignancy is found.

The reporter is of opinion that the condition he had to deal with was a glandular abscess, which from time to time evacuated itself, giving relief, only to fill up again and cause obstruction.

For a few days after the operation the patient was exhausted but there were no complications. She left hospital on July 21, 1925, and has remained well since and fit for her household duties.

Case II.—Papillomatous Mass Growing from the Bifurcation of the Trachea and Obstructing both Bronchi. Removed through the Bronchoscope. No Recurrence.—Mrs. C., aged 42, was admitted to a Medical Ward at the Western Infirmary, Glasgow, suffering from difficulty in breathing. As the physician could find no sufficient cause in the chest to account for the difficulty, she was sent to the Throat Department. Examination by the indirect method revealed a mass almost filling the larynx. With the aid of the suspension apparatus this was completely removed, the place of origin curetted, and 10 per cent. silver nitrate solution rubbed in. The mass was found to be papillomatous.

About a year later the patient was again admitted to the same medical ward with even more severe obstruction to the breathing. The services of the author were again requisitioned with the unkind hint that the former operation had not been efficient and successful. To his relief, however, the larynx was found to be quite clear and the patient was sent back to the physician. A more charitable feeling having been established a bronchoscope examination was suggested. This was carried out and a large mass was found, as far as could be seen completely blocking the entrance to the right bronchus and partially the left. This was removed with forceps and punch forceps, and was found to be growing from the bifurcation. The bronchial tree was examined for any growth at other parts, but with a negative result. The place of origin was well swabbed with silver nitrate solution. The pathologist reported that the growth was papillomatous. Since that time there has been no return of the difficulty in breathing and the woman is quite well.

Case III.—Suspected Foreign Body in Right Lung. Negative Result on X-ray Examination and on Bronchoscopy. A month later severe pain in front of, and below angle of Scapula where a patch of Surgical Emphysema developed. Miss M., aged 53. In June, 1925, she motored from London to Glasgow in an open car. Immediately after this she developed an irritating cough with severe spasms at times, but unaccompanied by expectoration and with no signs of hæmoptysis. She herself was under the impression that she had inhaled dust during the journey and that this was the cause of the cough. She was seen by a consulting physician who could find no cause for the cough, and thought the condition was a "neurosis." X-ray examination

104 Howarth: *Laryngeal Lesion with Apparent Miliary Tuberculosis*

of the chest showed some fibrosis of the lungs and possibly (?) slight dilatation of the aortic arch. Wassermann negative. No other evidence of aneurysm was found. Except for the cough she felt and looked quite well but the cough remained irritating in spite of treatment.

She was referred to the author on December 8, 1925. Beyond some congestion of the pharynx and larynx there was nothing abnormal. The cords moved normally. A bronchoscopic examination was carried out under chloroform. The bronchi and branches were examined in detail but nothing whatever was found. The cough persisted for a few days, but after a week disappeared and the patient left the nursing home. Four weeks later she was sent back to the home by her medical attendant, as she had developed a slight rise of temperature accompanied by sharp pain below, and in front of, the angle of the right scapula, where he also found a patch of surgical emphysema. The writer saw her again and there was no doubt about the emphysema. The chest was again X-rayed but again nothing was discovered. The emphysema and pain disappeared in a few days. The patient has remained in good health ever since, and has had no return of the cough.

The writer is puzzled as to the sequence of events, but suggests that the patient inhaled a small piece of flint or other sharp substance during her journey by motor-car, and that this foreign body found its way through the pleura (setting up a localized inflammation by which the pleural surfaces were united), and passing on to the subcutaneous tissue, where it remains at rest.

Discussion.—Sir JAMES DUNDAS-GRANT said that he had had a case resembling one of Dr. Syme's. The patient had had dyspnœa and stridor, chiefly inspiratory. With the bronchoscope a fimbriated body which moved with the breathing was seen at the bifurcation. He (Sir James) had removed this and the breathing had afterwards been perfect. One passage of the bronchoscope relieved the patient during a subsequent attack of dyspnœa. There was a syphilitic stricture of the trachea. The patient had improved greatly under appropriate treatment, and was allowed to go home, with the injunction to return if he had serious symptoms again. He did not return, however, until the dyspnœa was extreme, and on the night after his arrival he died.

Mr. RITCHIE RODGER said the moral of Dr. Syme's second case was that when laryngologists were asked for an opinion by their physician colleagues they should be careful to say they could not see anything with the laryngoscope, but that they would like to make a bronchoscopic examination. Two years ago he (the speaker) was asked to see a man in suffering from what appeared to be an attack of asthma. He could not find anything unusual in the larynx. A bronchoscopic examination was not made, and after death a papilloma just above the bifurcation was found, similar to that occurring in Dr. Syme's case.

Dr. SYME (in reply) said that at the Scottish Society, a year ago, he had pressed the point, as Mr. Fraser had done, that bronchoscopic examinations should be much more frequently carried out.

[*June* 10, 1927.

CLINICAL MEETING.

FURTHER REPORT OF CASE PREVIOUSLY SHOWN.[1]

Laryngeal Lesion Associated with Apparent Miliary Tuberculosis of the Lung.

By WALTER G. HOWARTH, F.R.C.S.

Mr. HOWARTH said that this patient was not now appreciably worse than when last shown, but the point of interest was the associated lung condition, and the analogy to the other case, the skiagram from which he had shown—that of the patient who had died from miliary tuberculosis in the lung. It was suggested that a blood-infection had already taken place, and that it was merely a matter of time for it to progress through the lung and for the tubercles to break down.

[1] *Proc. Roy. Soc. Med.*, 1927, xx (Sect. Laryng., 22).

Tumour of Naso-pharynx: Case for Diagnosis.

By WILLIAM IBBOTSON, F.R.C.S.

K. P., AGED 7.

History.—Makes noises when breathing during sleep. Persistent nasal discharge. Treated daily at his local hospital since August, 1926. Has recently received an external application of radium.

Examination, March 21, 1927.—Nose: Both nasal passages blocked with a hard suppurating mass. Naso-pharynx: Also filled by the above. Mouth: Downward bulging of soft palate. Transillumination: Frontal sinuses clear; right maxillary sinus clear; left maxillary sinus dim.

Operation.—The tumour was found to be very firmly fixed to the tissues forming the posterior choanæ, and though the naso-pharynx was completely cleared, it was found impracticable at this operation to attempt to remove the above fixed portion owing to the unsatisfactory condition of the patient.

Unfortunately the patient died from shock a few hours later.

Diagnosis.—It has been suggested that the tumour is of the nature of, (*a*) an odontoma, (*b*) a teratoma.

[Tumour itself and microscopic slides shown.]

Discussion.—Mr. BROUGHTON BARNES said he had had a case in a baby in which there was a skin-covered tumour in the nasopharynx, growing from the basisphenoid. It was readily removed with a diathermy snare and there was no hæmorrhage.

Mr. HAROLD BARWELL said that splitting the palate was not always the best way to reach these sessile nasopharyngeal tumours. Better access was to be obtained by lateral rhinoscopy.

Mr. IBBOTSON said that Mr. Layton had suggested to him that the tumour should be called epignathic teratoma.

Intralaryngeal Tumour in a Baby aged 14 Days.

By WILLIAM IBBOTSON, F.R.C.S.

FIRST seen on November 5, 1926.

History.—Crowing inspiratory stridor since birth; no v increasing, with cyanosis.

Operation.—Two hours later. Tracheotomy.

November 7, 1926.—Patient had progressed fairly well until this morning when he became deeply cyanotic and died.

Post-mortem Examination by Dr. Charles Sundell.—Heart: Healthy. Lungs: Patches of atelectasis in both organs. Thymus: Extremely large. Larynx: Upper aperture very narrow, with marked lateral incurving of both ary-epiglottic folds. A sprouting growth on each cord. Trachea healthy everywhere; no clot; no evidence of obstruction.

[The larynx was shown.]

Mr. IBBOTSON said it would be interesting to know if any Member had met with such a case at an earlier age.

Tumour of Frontal Bone.

By WILLIAM IBBOTSON, F.R.C.S.

N. H., MALE, aged 29.

History.—About eighteen months ago patient was admitted to hospital in the following condition: Completely unconscious; a large Pott's puffy tumour over the nasion; history of recent fits; history of an operation on the frontal bone in 1918.

106 Ibbotson: *Tumour of Frontal Bone;* Kisch: *Chronic Empyema*

Operation: Urgent. A large ivory exostosis was removed from the diploic tissue of the frontal bone, there being suppuration and definite pressure on the dura mater, which was not itself apparently traumatized in any way.

Patient recovered consciousness within twenty-four hours and made an excellent recovery.

May 19, 1927.—For several months past patient has consistently complained of "pressure" headaches, which occur with slowly increasing severity and frequency: there is also slight vertigo. General health good; he is able to perform his work.

Clinical examination and radiography suggest that a further exostosis is present.

[Tumour and radiographic films shown.]

Discussion.—Mr. RITCHIE RODGER said he was showing the skiagram and specimen of a recent case of his own. It was that of a woman aged 26, who had first come to hospital seven years ago, with a round, hard tumour in the ethmoid region, in the mid-line, obviously an osteoma. He had removed this growth by means of a horizontal incision, and after making preparations in anticipation of hæmorrhage, he found that one blow with the mallet dislocated the tumour, so that it was easily lifted out. He could see into the frontal sinuses, and both were normal. The wound healed by first intention, and the deformity disappeared. Two weeks ago she had returned with a complaint of considerable pain in the left temporal region, and some displacement of the left eye downwards with œdema of the upper eyelid. The temperature was 101° F. X-ray examination showed a dense, bony growth occupying the whole left frontal sinus, involving the left ethmoid, and projecting into the left antrum. He operated by making an incision as for the radical frontal sinus operation, and found that the tumour had filled the frontal sinus and eroded the inner table of the bone. The roof of the sinus was eroded to the extent of a square inch and the dura exposed, the whole tumour lying in pus. He then had to remove the inner wall of the orbit, which was involved in a dense, rounded, bony tumour mass extending downwards into the upper part of the antrum. The wound had healed by first intention, but there was considerable flattening of the left frontal region.

Mr. GRAHAM BROWN thought that this was a condition similar to ivory exostosis growing from the frontal sinus. The treatment was radical removal. He himself had had a case in which the tumour grew from the cavity of the frontal sinus, and appeared clinically to be an ethmoiditis, as there was a red swelling in the region of the inner canthus. A skiagram had shown the usual dense appearance. At operation an ivory exostosis was found protruding into the orbit, into the frontal sinus, which was much enlarged, and also back towards the sphenoid through the ethmoid cells. It had come away fairly easily, piecemeal. The muco-purulent discharge was confined to the frontal sinus; there was no evidence of discharge in the nose. The mucous membrane of the ethmoid region had not to be removed.

Cure of a Chronic Empyema of the Antrum, after Operation on the Frontal Sinus.

By HAROLD KISCH, F.R.C.S.

MRS. A. presented herself at hospital with a left-sided purulent discharge of the nose. On examination both antrum and frontal sinus contained foul pus, and the X-ray plate showed dense shadows. Operation on the frontal sinus was carried out by the method recently described by the author, and lavage of the antrum was subsequently performed. After three washings the antrum remained clear, and the patient is now quite well. The scar of the operation is practically invisible.

Discussion.—Mr. HERBERT TILLEY said that in many cases the antrum was merely the receptacle of pus, and its mucous membrane was not itself diseased. Some Members might remember a case he (the speaker) had shown many years ago, in which both frontal sinuses had been opened externally for chronic empyemata, but the antra had not been washed out after the operation, although beforehand they were filled with pus. He had seen the patient

about once a year since and had never observed any signs of antral suppuration. If the discharge from the upper sinuses was checked, the antra in many cases would take care of themselves.

Mr. T. B. LAYTON said he thought that this condition should be called pyosinus[1] to distinguish it from inflammation of the maxillary sinus. It was true that he did not know how to tell the one from the other, but the nomenclature was needed in order that the difference might be borne in mind. Thereby we might learn to make the diagnosis in the future.

Dr. W. S. SYME asked whether Mr. Tilley deliberately used the word "many." He hoped not, as his own view was that there were not many cases in which the antrum was merely filled with pus from the frontal sinus.

Mr. HERBERT TILLEY (answering Dr. Syme) said that perhaps he had used the term "many" somewhat loosely, but during thirty years he had seen a number of such cases, in which the purulent discharge in the antrum cleared up after the frontal sinus suppuration had been cured. The cases were parallel with those in which pus was passed with the urine. This might come either from the kidney or the bladder, or from both, but if only from the former the "purulent cystitis" might cease when the renal lesion was cured and without any direct treatment of the bladder.

Mr. SOMERVILLE HASTINGS said Mr. Kisch was to be congratulated on the result in this case, as it was an open question whether this was a justifiable operation. Many years ago he had seen two cases in which the ethmoidal region was operated upon in the presence of an antrum containing foul pus. Both the patients had died from meningitis, and the fact made a profound impression upon him (the speaker). In his opinion it was very doubtful whether one was justified in doing anything operative to the upper region of the nose when there was a definite reservoir of pus in the antrum. He did not think a cavity like the antrum could contain pus without having some infection of its mucous membrane. His practice was to abstain from any operative procedures in the upper regions of the nose when the antrum was full of pus. In the present case he would have dealt with the antrum first, leaving the frontal sinus until later.

Mr. KISCH (in reply) said he agreed with most of the remarks that had been made. The difficulty was to determine beforehand whether the antrum was acting merely as a cistern, or whether it was actively diseased. Before doing an antrum operation he made it a practice to try to ascertain whether the frontal sinus was diseased. For that reason he had all the sinuses examined by X-rays. If the frontal sinus was implicated, he operated upon that first; he did not see how one could expect to get a successful antrum result if the frontal sinus was diseased. He washed out the antrum beforehand. During the operation he was careful to avoid unnecessary damage to the bone, therefore he did not use burrs or rasps. Then, he thought, there was no extraordinary risk to the patient.

Case of Enlargement of the Frontal Bone, Maxilla and Mandible.

By T. JEFFERSON FAULDER, F.R.C.S.

MRS. W. H., aged 26. Enlargement (unilateral right) of frontal bone maxilla and mandible. Right nasal obstruction.

The growth had ceased when the patient was aged 21.

[Skiagrams shown.]

Mr. W. M. MOLLISON said that the skiagram was much like that of Paget's disease.

Mr. NORMAN PATTERSON said he had seen this patient seven years previously, and the growth had not altered since then.

[1] After Hajek (*vide* "Nasal Accessory Sinuses," 1926, i, 230).

Three Cases of Epithelioma Treated Successfully by Deep X-rays.

By W. M. Mollison, F.R.C.S.

Case I.—Patient, male, aged 54, seen in September, 1926, on account of "sore throat" of twelve weeks' standing. There was a large epitheliomatous ulcer on the left side of the back of the tongue extending as far as the epiglottis.

The ulcer had the typical hard raised edge of epithelioma. The patient had been advised to have his tongue removed.

Diathermy was carried out in October, 1926, but on account of the position of the ulcer great difficulty was experienced; indeed it is doubtful whether any good was done, as on December 15 the ulcer had become larger.

Dr. Watt gave a series of doses of deep X-rays in November, 1926, and again in December, 1926.

It will be seen that the tongue now presents a smooth, almost normal, surface over the site of the previous ulcer. Has gained 5½ lb. in weight.

Case II.—Patient, male, aged 54, was seen in January, 1923. There was a large epithelioma involving the posterior part of the tongue, and the epiglottis; it was so extensive as to overhang the larynx. Diathermy was applied. Dr. Watt then gave two series of doses of deep X-rays, October, 1923 and January, 1924. Last dose, a single one, in July, 1924.

Examination now shows a smooth, supple scar at the back of the tongue. Patient has put on 2 st. in weight.

Case III.—Patient, male, aged 52, was operated upon three years ago at Guy's Hospital for growth of the right antrum and ethmoid; section showed squamous epithelioma.

A further operation for recurrence was performed and finally sixteen months ago the right eye was removed on account of extensive orbital involvement. Dr. Watt gave five series of doses of X-rays—the last fifteen months ago. The patient has put on 2½ st. in weight and is back at work.

Sarcoma of the Tonsil treated by X-ray Therapy and Excision.

By C. Gill Carey, F.R.C.S.Ed.

Mrs. C., seen on March 31, 1926, complaining of swelling in the throat of six weeks' duration.

Examination showed a large, firm mass, without ulceration on its surface, occupying the right tonsillar fossa. There were no palpable glands in the neck.

As the patient was then eight months pregnant, it was decided to postpone operation until the child had been born. The growth increased so rapidly in size, however, that serious dyspnœa occurred, and the patient was shown to Dr. W. L. Watt, who treated her with deep X-ray therapy on three occasions between April 9 and 13. This treatment reduced the size of the tumour sufficiently to allow her to have a normal labour on April 15.

Five days later the tumour was removed through the mouth. Sections cut from it were reported on as sarcoma.

Following the operation a second series of three radiations was given.

Discussion.—Dr. Andrew Wylie (President) said that Mr. Mollison's cases were so successful that it appeared as if in the future surgery in this region would be replaced by the use of radium.

Mr. NORMAN PATTERSON said he was much impressed with these results, and asked whether in each case a specimen had been taken for microscopical verification. It would be interesting to know whether or not there was a tendency to differentiation of cells.

Mr. A. R. TWEEDIE asked for further details as to the treatment.

Dr. JOBSON HORNE asked how many attendances on the part of the patient the deep X-ray course necessitated.

Mr. MOLLISON (in reply) said that the first case which drew his attention to the enormous value of deep X-ray therapy was that of a woman whom he had seen four years ago, because she was suffering from a post-cricoid carcinoma; ulceration could be seen above the arytænoids, and she was only able to swallow water. Dr. Finzi applied X-rays deeply; three weeks later she could swallow fluids comfortably, and six weeks later she was able to swallow almost anything. She was still alive and well. He then decided to employ this method in all cases in which recurrence was probable and the approach to the region was very difficult. Dr. Watt, of Guy's Hospital, was an enthusiast in this matter, and at his own cost he had supplied to the hospital a deep X-ray apparatus. He (the speaker) had asked Dr. Watt to treat his cases of this kind. Members of the Section knew the brilliant results obtained by Mr. Norman Patterson by means of diathermy, but, in his own hands, diathermy had not been a success for growths in difficult situations.

In only two of the cases were microscopical sections taken. In the first case it seemed so obvious that the growth was carcinoma that it did not seem necessary to take a section. In the second case, growth of the epiglottis and the deep pharynx, a section was taken, and showed squamous-celled carcinoma, which was the nature of the growth in the case of the man whose orbit had been exenterated.

He could not give Mr. Tweedie the details as to the tubes used; he hoped Dr. Watt would read a paper on the subject before the Section during next Session. Dr. Watt usually gave the treatment from forty to sixty minutes three times in the first week, aiming the rays at different portions of the neck. If the result seemed to be good, an interval of a month or two months was allowed, then another series of doses was given, of much the same duration. He (the speaker) thought that the results in cases of sarcoma were more striking than those in cases of carcinoma. For example, he had seen, fifteen months ago, a patient who appeared to be in the last stages of sarcoma of the naso-pharynx. He had suggested treatment by deep X-ray, which was given by Dr. Carter Braine, Dr. Watt's assistant, and in a week that patient was practically well. Dr. Braine, however, thought there would be recurrence in a short time, and sent the patient back to his home in South Africa, whence letters later arrived from his friends stating that no disease could be seen, and indeed doubting the previous presence of growth. The man had remained free from disease for a year.

Papilloma of Stenson's Duct followed by Great Enlargement of Parotid Gland: Case for Diagnosis.

By H. NORMAN BARNETT, F.R.C.S.

H. D. B., AGED 71, male. First seen last year suffering from rheumatism and complaining of pain in the throat of a year's duration.

On Examination.—Tonsils unhealthy. Papilloma of Stenson's duct (left). Throat and mouth were treated and patient rapidly improved. Returned later with ulcerated surface of the hard palate. Ultra-violet rays now used for the throat and surface of the palate; condition temporarily improved but subsequently became worse. Patient underwent radium treatment, which had the effect of improving ulcerated surface. Some months later his doctor reported that he had acute illness, the nature of which he did not know. Symptoms pointed to mumps. When seen again there was found to be papilloma of Stenson's duct on right side with great enlargement of the parotid gland, the bony outlines of the jaw being completely obliterated with enlargement of the cervical glands on the same side. Under treatment the condition greatly improved, the parotid became normal and the glands smaller. The ulcer in the

110 Barnett—Carruthers—Gibb

mouth, however, began to increase and radium treatment was again given but there was no improvement. Subsequently the glands on the right side again became very much enlarged and those on the left began to increase in size. A short time afterwards a gland on the right side suddenly broke down and operation was performed, when a large pus cavity was found and drained, and the broken-down gland removed. *Pathologist's report:* "Pus contains streptococcus; a scraping from the ulcer of the palate showed streptococcus in short chains and degenerate epithelial cells, no tumour cells in the spreading margin. Blood-count normal; urine normal; Wassermann negative."

Discussion.—Dr. W. HILL asked whether the treatment had been by ultra-violet rays only.

Sir JAMES DUNDAS-GRANT asked whether examination had been made of the piece from the ulcer of the hard palate. It looked like epithelioma, with secondary involvement of glands on both sides.

Mr. NORMAN BARNETT (in reply) said that the papilloma had been removed, and ultra-violet rays and radium applied. No tumour cells were found in the growth nor at the spreading edge. He did not think the case was so simple a one as was suggested. There was especial difficulty in explaining the extreme swelling of the parotid gland, which subsequently subsided, and the presence of a large amount of pus in the neck containing streptococci.

Suspected Papilloma of the Larynx: Case for Diagnosis.

By N. STUART CARRUTHERS, F.R.C.S.

A. B., AGED 27. First seen July, 1926. Patient complained of hoarseness of seven months' duration. Examination revealed what appeared to be a papilloma in the anterior commissure and growing from the right cord.

August 6, 1926.—The growth was removed through a laryngoscope and the base cauterized.

March 23, 1927.—Patient re-admitted to hospital; the papilloma, which was as large as on the previous occasion, was removed and the base again cauterized.

Pathological Report.—"This tissue is of an epitheliomatous nature."

Present Condition.—There is a large papillomatous mass in the anterior commissure growing from the right cord, but there is also some infiltration of the left.

[Microscopic section of growth shown.]

Paralysis of Right Vocal Cord: Case for Diagnosis.

By J. A. GIBB, M.D.

MRS. Y., aged 23.

History.—Two years ago had greatly enlarged glands in her neck; was treated in a sanatorium. Glands incised; discharged for twelve months. On healing, voice became husky. There was never any cough and no sputum. No pulmonary signs.

Examination of larynx shows right vocal cord paralysed in position of extreme abduction.

Case for Diagnosis.

By C. HAMBLEN THOMAS, F.R.C.S.

F. S., AGED 60, male. Occupation, instrument maker.

History.—Six months ago began to have attacks of coughing, worse at night, with associated swelling at root of neck (? emphysematous). Lately huskiness of voice. No dyspnœa. No dysphagia. Not losing weight. No symptoms referred to chest.

Examination.—Pharynx: chronic tonsillitis; papilloma on uvula. Larynx: left vocal cord showed a pale limited smooth swelling anteriorly; no ulceration; cord somewhat limited in movement. Neck: fullness above clavicles; no definite glandular enlargement. Chest: nothing abnormal seen by X-rays or clinically. Sputum and blood not examined.

Treatment.—Vapours and sedative cough mixture. Slight improvement.

Discussion.—Sir STCLAIR THOMSON said that Mr. Carruthers' case might possibly be one of malignant disease. These growths were more common in the anterior third of the cord than in the posterior. His (the speaker's) youngest female patient was aged 33, and the disease in her case was of very malignant type. Recurrence had been treated by hemi-laryngectomy, and the patient was still well seven years after the operation. He suggested that a further piece of the growth should be removed and examined. If it were found to be epitheliomatous, the case should be dealt with by laryngo-fissure. The greater part of the right cord might have to be removed, and half the left. In some cases, if there was stenosis, the patients managed very well with a tracheotomy tube. The idea of laryngectomy should not be entertained.

With regard to Mr. Gibb's case, it was difficult to say whether there was anything subglottic.

Mr. HERBERT TILLEY said he did not think it was correct to speak of Mr. Gibb's case as one in which the "right vocal cord was paralysed in a position of extreme abduction." The cord was not paralysed; the arytænoid moved with comparative freedom. There was some ulcerative condition of the cord which, in parts, gave it a congested granular appearance.

Professor GEORGES PORTMANN (Bordeaux) said he did not regard Mr. Gibbs' case as one of paralysis of the vocal cord. He thought the patient had tuberculous trouble, and that the vocal cord and the false cord were pulled out by cicatricial tissue.

Mr. H. SMURTHWAITE said that there was scar tissue in this case which he was sure was one of tuberculous disease of the cord. There was hoarseness, but patients with one cord paralysed could always produce some voice. One could only see a thin streak of the right cord, and it was partially fixed.

Sir JAMES DUNDAS-GRANT said that a paralysis starting in the right vocal cord was comparatively rare, while paralysis of the cord in extreme abduction was almost unknown. The cord was in the position of extreme abduction and was immobile, but in his opinion the immobility was due to local cicatricial contraction, not to involvement of the laryngeal nerve. There was probably a mitigated form of tuberculosis, which had arrived at a stationary condition.

Dr. J. S. FRASER said he considered that Mr. Carruthers' case was one of malignant disease of the vocal cords. Not only was the right cord affected, but the condition had spread round the anterior commissure to the anterior part of the left cord. He agreed that in young people the malignancy of these growths was high. Therefore he was surprised to hear Sir StClair Thomson recommend laryngo-fissure in this case; he would have thought it more suitable for laryngectomy.

Dr. ANDREW WYLIE (President) said that with regard to Mr. Carruthers' case, a piece of the growth should be removed and examined under the microscope. The condition in Mr. Hamblen Thomas's case had a tuberculous appearance.

112 Gibb: *Sinusitis with Complications; Frontal Sinusitis*

Mr. CARRUTHERS (in reply) said that on the first occasion the growth in this case was completely removed, and its base touched with the cautery. After two months it began to grow again. The second removal was by the same method—direct laryngoscopy. The growth had a broad base and he removed it as far as possible. It rapidly recurred. The pathologist reported it as an epithelioma, but would not be definite on this point.

Mr. GIBB (in reply) said that in his case there had never been any sputum or evidence of active tuberculosis. The patient had entered a sanatorium because of a large number of diseased and discharging cervical glands. She had been in the sanatorium two years. Her voice was unaffected during that time and while the glands were discharging, but as soon as healing took place loss of voice had occurred. Consequently, it occurred to him that the laryngeal nerve had been caught up in scar tissue. He was willing to accept the opinion of more experienced Members with regard to the cord. No reaction had been obtained on stimulating the laryngeal nerve. He proposed to tell the patient that nothing further could be done for her.

Case of Sinusitis with Complications: Phlegmon of Orbit.

By J. A. GIBB, M.D.

J. W., AGED 2.

November 12, 1926.—Admitted to hospital with redness and œdema of both upper eyelids and profuse discharge of pus from both nares.

Operation.—Right and left maxillary antra opened. Right ethmoid region freely opened.

Temperature between 100° and 102° F. developed, and on November 22, 1926, a fluctuant swelling at the right inner canthus was opened and pus evacuated.

November 24, 1926.—As there was evidence of pus in the orbit an incision was made through the lower lid and a good deal of pus was evacuated. The wound was gently scraped and drained by a wisp of gauze.

November 26, 1926.—Discharged to the out-patient department.

Case of Frontal Sinusitis.

By J. A. GIBB, M.D.

R. S., AGED 22.

Consulted me on March 22, 1927, complaining of frontal headaches. Headaches marked on rising in morning and usually lasted until 11 or 12 o'clock. Pressure over right supra-orbital nerve and at inner angle of orbit caused pain. Pus in mid-space.

Transillumination.—Right frontal sinus dark, and maxillary antrum dark.

Admitted to nursing home next day.

March 24, 1927.—Operation: Small incision at inner angle of orbit, anterior wall of frontal sinus removed; sinus full of pus; carefully swabbed out. Anterior half-middle turbinate removed and fronto-nasal duct enlarged by burring; tube inserted into nose.

March 29, 1927.—Tube removed and nares douched.

April 8, 1927.—Owing to swollen condition of wound and rise of temperature to 104·8° F. the original incision was carried to the full extent of eyebrow, and anterior wall of sinus completely removed as well as part of floor. Direct communication with the nasal cavity was made by burring; wound sutured, with exception of outer portion, which was drained.

April 10, 1927.—Definite attack of erysipelas developed. 20 c.c. anti-streptococcus serum given at twelve-hour intervals for three days, when calcium chloride and iron and quinine were administered.

April 15, 1927.—Temperature normal.

April 29, 1927.—Left nursing home quite well.

Case is shown with a view to elicit discussion as to the value of the small external incision.

Discussion.—Dr. WYLIE (President) said that in the second case the patient seemed to be quite cured; he could not detect any pus.

Sir JAMES DUNDAS-GRANT said that Mr. Gibb was to be congratulated on his result in this difficult case.

Mr. GIBB (in reply) said he had two patients in hospital, one a boy, aged 10, the other a younger child, both suffering from ethmoiditis. He performed an external operation on the first of these, and healing occurred without complications. In the second case he operated by the external route; pus appeared at a later stage. He thought that in these cases the operation should be performed externally.

Cases Illustrating Mr. T. B. Layton's Paper on "Nasal Polypi."[1]

I.—L. M., GIRL, aged 15. Polypi both sides of nose, often removed without effect. Much pus each side of nose, but after repeated examinations I could not persuade myself that she had suppurative sinusitis anywhere. X-ray examination: Ethmoids and perhaps sphenoids foggy. Operation January 7, 1927, under intra-tracheal ether: removal of ascending process of superior maxilla and lachrymal bone. Anterior ethmoidal cells contained gelatinous material. All anterior ethmoidal cells removed lateral to mid-turbinal. One posterior ethmoidal cell cleared out.

II.—Elderly man. After extraction of all teeth had "a full feeling" right side of head. Single polyp right side, no pus. Antrum opened January 14, 1927, under local anæsthesia. No secretion, no discoloration, no general swelling of mucous membrane, some rather large vessels. At first I thought it was normal, then I noticed certain areas whiter than the rest. I touched one of these and it burst, exuding a bead of pus. I then realized that the mucous membrane was studded with twenty or thirty such areas, each the size of a sago grain. From swab from pustule nothing was grown except what were thought to be organisms of an air-borne contamination. Polyp removed, ethmoid explored through antrum, apparently normal, all mucous membrane curetted away and radical maxillary operation (Denker) completed. Patient still complains of "full" feeling.

III.—G. E. B., male, aged 54; had been advised to have tonsils removed for rheumatoid arthritis.

December 13, 1926.—Polyp, left side, removed; both antra washed out; nothing found.

December 31, 1926.—Suspicion of muco-pus over inferior turbinal, below orifice of antrum on each side.

January 3, 1927.—Posterior rhinoscopy (poor view); suspicion of pus above mid-turbinal on right side. Maxillary sinuses washed out—right; left, suction with syringe a few flakes—fluid injected *nil*, on blowing air through to empty sinus some pus blown into meatus. X-ray. Operation, local anæsthesia. Left side—polypoid condition, especially at bottom, flaky purulent secretion, most

[1] See p. 81.

of mucous membrane scraped away. Right side—upper part apparently normal; lower part, commencing polypoid condition. This scraped away, the rest left.

IV.—Mrs. B., middle-aged. Polypi each side of nose; pus in each antrum.

First operation: Local anæsthesia; bilateral radical operation on maxillary—pus in cavity; mucous membrane inflamed, not swollen; tooth removed—cultivation gave a mixed growth of *Staphylococcus albus*, *Streptococcus longus* and torulæ; swab from antrum, *Staphylococcus aureus*.

Second operation: Intratracheal ether, all ethmoid on each side removed lateral to middle turbinal through external incision. All cells full of polypoid mucous membrane, but *no* pus.

Aneurysm of Internal Carotid.

By E. Broughton Barnes, F.R.C.S.Ed.

T. O. C., MALE, aged 71. First seen on April 5, 1927, complaining of slight dysphagia of two years' duration. Internal carotid aneurysm, best seen with laryngeal mirror.

No abnormal physical signs in the chest.

Discussion.—Professor Georges Portmann said that four years ago he saw a similar case, which he published in the *Archives médicales Belges*. He had seen very few cases of aneurysm of the carotid artery. Differential diagnosis was sometimes difficult in cases of aneurysm of the pharyngeal artery.

Mr. Hamblen Thomas did not think this was aneurysm of the internal carotid. Often the large vessel seen in that situation was the ascending pharyngeal. The carotid tended in that direction when the head was flexed, and it was the bend which caused visible pulsation and led to the mistaken diagnosis of aneurysm.

Dr. W. Hill asked what was the objection to trying ligature?

Mr. H. Tilley said the objections to ligaturing the artery were the man's age, and the fact that he had had the condition a long time and was fairly comfortable.

Dr. Andrew Wylie (President) said he did not consider that the swelling was an aneurysm; it was probably a dilatation of the vessel.

Mr. Broughton Barnes (in reply) said he did not follow Mr. Hamblen Thomas as to the reason of the curve. This was not a saccular aneurysm. He considered there was a fusiform expansion. He agreed that tying the vessels should not be done, because of the patient's age and the probable condition of the fellow vessel, which might not give an adequate blood-supply to the brain.

Case of Fracture of Larynx.

By T. B. Jobson, M.D.

M. F., FEMALE, aged 26. Injured in motor accident on May 30, 1927; struck on left side of neck; she coughed up some blood during next twenty-four hours. There was pain in the throat, with dysphagia and dysphonia; surgical emphysema over larynx, June 10, 1927. Crepitus could still be felt; outline of cords was irregular and larynx still showed evidence of trauma; there was no paresis present.

Discussion.—Dr. SALISBURY SHARPE said there was stiffness in the crico-arytænoid joint. A somewhat similar effect had ensued in a case which he had had thirty-nine years ago, of comminuted fracture of the thyroid cartilages. Two years after the accident the patient had had a very good voice, and the only abnormality was a slightly lessened amplitude of the crico-thyroid movement on one side; this would probably follow in the present case.

Sir JAMES DUNDAS-GRANT said this was a remarkable recovery from a dangerous accident. Sir Felix Semon had once shown a case in which a man almost lost his life after such an accident, which had occurred during a game of football.

Mr. JOBSON said that he had seen the patient for the first time on the previous day. Such cases were more frequently seen in the days when garotting was a common crime. This patient's symptoms were now steadily subsiding. He wondered whether he ought to strap the larynx, as it was still crepitating. He proposed to enjoin silence on the patient.

Chronic Enlargement of the Upper Jaw.

By G. W. BADGEROW, C.M.G., F.R.C.S., and F. C. ORMEROD, F.R.C.S.

MRS. A. B., aged 65. For six years has noticed an enlargement of the left side of the face. The extent of the swelling has varied from time to time, but latterly has been fairly constant.

A number of carious teeth had been removed shortly before the onset of the swelling; others had been falling out since she was 17. (Evident history of pyorrhœa.) Attacks of pain in face from time to time.

The points of special interest are: (1) The massive enlargement of the maxilla, including the alveolar margin and its extension on to the nasal process and to the opposite side of the nose; and (2) the encroachment on cavity of maxillary antrum by thickened walls.

Fibromata (or ? Papilloma) of Larynx.

By W. H. JEWELL, M.D.

T. F., MALE, aged 37. "Losing voice" for five months.

There is a rounded tumour about the size of a pea attached to the anterior margin of the right vocal cord. Below and in front of this nodule there is another one which is not so definitely circumscribed.

Postscript: The tumours were removed by means of Haslinger's directoscope, and the patient has recovered his voice and is able to sing.

The pathologist reported that the growth attached to the vocal cord was a soft fibroma and that the other one was suggestive of an epithelioma, but admitted it might have been due to the way in which the section was cut. No recurrence two months later.

Report of Section of Laryngology Research Committee.

Mr. BELL TAWSE'S case of "Perforation of the Palate,"[1] shown at Meetings of the Section on December 4, 1925; June 4, 1926; and November 5, 1926, was considered by the Research Committee, on May 12, 1927.

The microscopical section of a portion of the growth removed was submitted to Mr. T. W. P. Lawrence, F.R.C.S., and Dr. G. W. Nicholson, as Referees, and they report as follows:—

[1] *Proc. Roy. Soc. Med.*, 1926, xix (Sect. Laryng., 23, 26); 1927, xx (Sect. Laryng., 2).

116 *Report of Research Committee*: Irwin Moore: *Œsophagectasia*

The section examined shows downgrowths of columns and tubes—lined with a single layer of cubical cells—into the deeper tissues. These can be traced to the surface epithelium, which is ulcerating and granulating. It is a basal-celled carcinoma (rodent ulcer).

(T. W. P. Lawrence, F.R.C.S.)

The tumour is more tubular in structure than the average rodent ulcer of squamous epithelium. Parts of it bear a close resemblance to salivary gland tumour. It is therefore possible that the tumour arose in connexion with mucous glands. This does not, however, alter the fact that it is a basal-celled carcinoma.

(G. W. Nicholson, M.D.)

The Pathology of Œsophagectasia (Dilatation of the Œsophagus without Anatomic Stenosis at the Cardiac Orifice).[1]

Note by Dr. Irwin Moore.

Since publication of this paper in the *Proceedings* Dr. Arthur F. Hurst has referred me to his paper "The Sphincters of the Alimentary Canal and their Clinical Significance," published in the *British Medical Journal*, 1925 (i), 145, in which it appears he first suggested that some such lesion would probably be found—as proved to be the case within a few months in three cases investigated pathologically at Guy's Hospital: one by G. W. Rake, who demonstrated well-marked degenerative changes, and in early cases active inflammation in the lower end of the œsophagus, particularly involving Auerbach's plexus; and in the second and third specimens by Professor Adrian Stokes, who found that the whole of the nerve plexus in the neighbourhood of the cardiac sphincter had been destroyed, and was replaced by fibrous tissue. One of the latter specimens (Grey Turner's case) I have described on p. 40.

In carrying out my investigation I was unaware of the opinion suggested by Dr. Hurst, and of the work of Professor Stokes and Dr. Rake.

1 See *Proceedings*, 1927, xx (Sect. Laryn., 31-41).

Section of Odontology.

President—Mr. J. B. Parfitt, L.R.C.P.Lond., M.R.C.S., L.D.S.Eng.

Further Investigation of the Pathology of Dentigerous Cysts with a New Treatment Based Thereon.

By Evelyn Sprawson, M.C., L.R.C.P.Lond., M.R.C.S., L.D.S.Eng.

Five years ago[1] I put before you a case of multiple dentigerous cysts and some remarks on what I considered to be the probable pathology of such cysts.

I gave ten reasons in support of the hypothesis then put forward and supported it in so far that I was able to show a granuloma on a deciduous tooth, part of a dental cyst attached to a deciduous tooth, to quote another case in support, and to demonstrate three of the other points; five others of the remainder were already recognized clinical facts. Since then I am not aware that any adverse

Fig. 1.—Granuloma on deciduous tooth.

Fig. 2.—Granuloma on deciduous tooth.

criticism has been made concerning the views I then advanced, nor have I yet heard of a case of a dentigerous cyst in man involving a tooth, the deciduous predecessor of which was *in situ* and alive.

To-night I show you some further specimens bearing on the same hypothesis, and put before you a form of treatment for these cysts which, under favourable circumstances, seems to me to be an improvement on what has been done for them in the past. I will now refer to the new specimens bearing on my last paper, which are here on exhibit.

Exhibits 1, 2 (fig. 1), 3 (fig. 2), 4, 5, 6, 7 and 8 are a series of specimens of granulomata *in situ* on deciduous teeth; on the fourth specimen there may indeed be

1 *Proceedings*, 1922, xv (Sect. Odont.), 56.

68 Sprawson: *Investigation of the Pathology of Dentigerous Cysts*

two small cysts, one at each apex. Since I read my last paper before you I have met with many such, some of the best of which are those on view; they help to bear out my contention that the sequelæ of caries in deciduous teeth are in no way different from those occurring in permanent teeth, but are less often seen because of the much shorter time that the deciduous teeth are present in the jaws.

Two other specimens (exhibits 9 and 10), though they have no connexion whatever with cysts, also bear this out; they are specimens of productive pulpitis in deciduous teeth, commonly known as "polypus" of the pulp. This, I believe, is a much rarer condition than granuloma in the deciduous dentition.

In 1922 I also stated that the only histological difference between the granulomata on deciduous and permanent teeth which I had noticed was that in the former

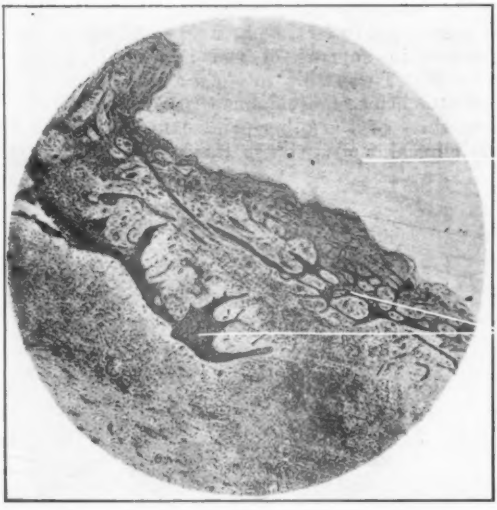


FIG. 3.—Photomicrograph of section of granuloma on deciduous tooth *in situ*.

there were much grosser masses of epithelium, and I accounted for that by reason of the greater vascularity of that part during the active tooth forming and tooth absorbing period and the greater youth and activity of the epithelial cells involved. Of course one meets with masses of epithelial cells in connexion with most granulomata on permanent teeth, but there does not seem to be anything like the same exuberance of epithelial cell growth.

The following exhibits, 11 (fig. 3) and 12, well illustrate this point, and I might add that I have never yet met with a granuloma on a deciduous tooth which did not show such epithelial masses. I have now examined about twenty of these granulomata; of course only a small portion of the epithelium can be shown in the photomicrographs; one needs to see the slides on exhibit and move them about, in order to appreciate this profusion of epithelium fully. I would here like to mention that one text-book referring to my past paper on this subject denies the existence of

granulomata on deciduous teeth, and remarks that a dentigerous cyst which involved a third molar did not seem so feasibly to have origin from a cyst on a deciduous tooth. I made the same remark myself in that paper and mentioned that though this was possible owing to the fact that cysts may burrow considerably, particularly when they reach the medullary portion of the bone, the first permanent molar (which is given as the commonest region in which dental cysts occur) was possibly, not to say probably, the most frequent source of origin of the cyst in such cases. Though the involvement of the third molar in a cyst is apparently not very rare, it has not been my good fortune yet to meet with such a case.

The next slide (exhibit 12A, fig. 4) is from the only skiagram I have seen showing the mandibular third molar involved in a cyst, and this slide confirms the probability of my suggestion; the cyst in this case most likely originated from the missing first permanent molar; I have no knowledge as to whether the second permanent molar,

FIG. 4.—A mandibular third molar in a cyst. The first permanent molar is missing and the second permanent molar filled.

which is *in situ* and filled, was a live or dead tooth, but the cyst might alternatively, but not so probably, have originated from that tooth.

I also suggested that "there would seem to be a race between the growing cyst to envelop the permanent tooth and the tooth to escape its obstruction and erupt," and the next specimen I have to show (exhibit 13) has a bearing on this, but is a doubtful one as I was unable to make a microscopical examination.

This is a case in a female aged $10\frac{4}{12}$ years of what is apparently a cyst (exhibit 14) in the premolar region, the commonest region for such (dentigerous) cysts. This cyst has become disconnected with the deciduous tooth which probably gave origin to it by that tooth being shed; the succeeding permanent teeth were able to avoid "end on" contact with the cyst in the process of eruption, so that the cyst here appears entirely disconnected with the permanent teeth and outside them.

Such cases have been recorded previously by others and by myself and confirmed by microscopical examination.

70 Sprawson: *Investigation of the Pathology of Dentigerous Cysts*

I am also showing another specimen (exhibit 15, fig. 5) together with a photomicrograph of a portion of it (exhibit 16, fig. 6), of a deciduous tooth with part of a cyst wall adherent to it, almost exactly similar to the photomicrograph I showed in 1922, except that this is larger and perhaps more diagrammatic; and in addition a photomicrograph of a small complete cyst (exhibit 17, fig. 7) with an entire epithelial lining, attached to the root of a deciduous tooth.

FIG. 5.—A deciduous tooth with a portion of a cyst wall attached.

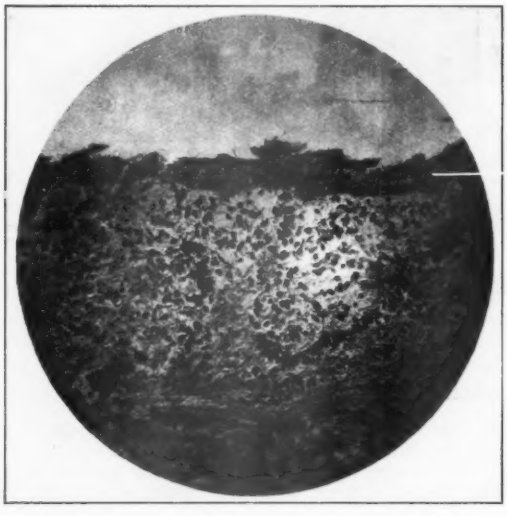


FIG. 6.—Photomicrograph of portion of cyst wall of fig. 5.

I now come to the cases, four in number, which have been subjected to a form of treatment the successful result of which is entirely dependent on the correctness of the hypothesis that the cyst is merely the obstructing agent to the eruption of the underlying permanent tooth. This underlying permanent tooth, not being readily absorbed, tends to become surrounded by the cyst as it grows.

The new features of this operation consist in the preservation of the tooth involved in the cyst and in the retention of a considerable portion of the cyst lining. Thus the operation is simplified into opening the cyst cavity freely enough to merge it into the buccal cavity, drainage thus becoming almost automatic and lavage being made easy to the patient. The cyst lining is not removed because it is epithelial and therefore protective. If it were removed there would be considerable risk of damage to the permanent tooth concerned which it is desirable to conserve; there would be also a very much larger raw surface open to infection and the absorption of toxins after operation; there would be more hæmorrhage and more pain. After all, there does not seem to be much object in removing one epithelial lining when the desirable ultimate result is that another epithelial covering should grow in from the edges of the wound and replace it. Moreover the operation is thereby rendered much simpler and shorter and the wound does not need any packing in order to arrest hæmorrhage. I am aware, of

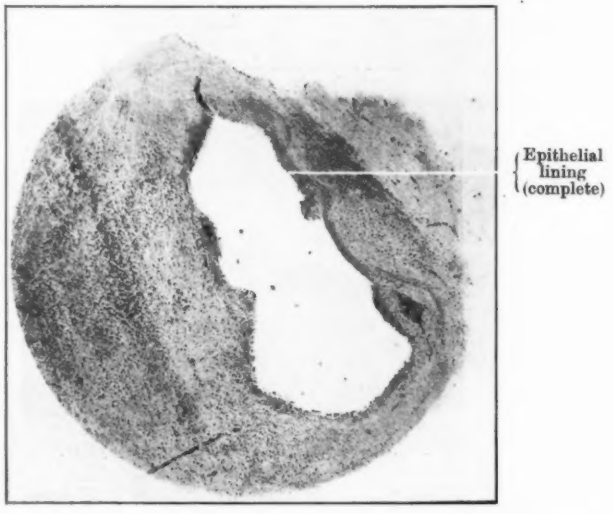


FIG. 7.—Photomicrograph of small entire cyst *in situ* attached to part of a deciduous tooth.

course, that a similar retention of cyst lining is occasionally practised when treating dental cysts. This treatment conserves the permanent tooth and you will see skiagrams, serial models and photomicrographs of sections from each case quoted.

The four cases I am showing were all operated upon under local anæsthesia.

Case I.—J. H. (exhibit 18), a male, aged 9, presented himself on January 10, 1925, with a painless and non-discharging swelling in the mandibular right deciduous first molar region, that tooth being present, carious, and having a dead and infected pulp. A skiagram was taken (exhibit 19) which showed considerable rarefaction of bone round the upper portion of the underlying first premolar. The rarefied area has not the sharp outline usually associated with a non-infected cystic swelling, and the same is true of two of the other cases I am showing, in both of which the cysts were small (Cases III and IV). This is possibly accounted for by the fact that its size had not yet reached that stage when surrounding tissues have been acted upon long enough to form a really sharply defined outline.

72 Sprawson: *Investigation of the Pathology of Dentigerous Cysts*

The deciduous first molar was removed and a piece of tissue was removed and placed in 5 per cent. saline formalin.

When this had been done the only attachment of the underlying premolar was by the pulp or dentine papilla at its base; it was very freely mobile and it seemed as if a sharp jet of water from a syringe might easily wash the tooth free from its attachment, almost the whole length of the tooth being exposed to sight. The tooth was left alone, a mouth wash prescribed and the patient instructed to use only the other side of his mouth.

The piece of tissue removed shows on microscopical examination (exhibit 20) one of the common appearances seen in the walls of such cysts, namely a lining of epithelial cells of varying degrees of depth, from two to fifteen cells deep, and in places giving rise to processes of epithelial cells which occasionally loop over and join on the outer aspect. Outside this was a somewhat thick layer of connective tissue forming a loose capsule, and external to that was a layer of thinned out alveolar bone.

In the few days subsequent to the operation the adjacent soft tissues closely invested the exposed premolar, so that even the point of a cusp was hardly to be seen. Twenty-seven days after operation it seemed safe to take an impression which (exhibit 21) shows the tip only of the larger cusp presenting at the bottom of the depression.

The subsequent history of the case is illustrated by the three following exhibits (22, 23 and 24) made respectively 124, 171 and 293 days after operation, the final one showing the

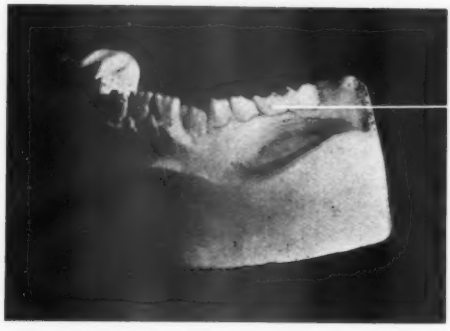


FIG. 8.—*Case II*, E. C., male, aged $9\frac{1}{12}$ years.

tooth fully erupted and in its normal position in the arch; it was not yet in occlusion, as the corresponding maxillary teeth were then only commencing to erupt, but it has since come into normal occlusion.

Case II.—E. C. (exhibit 25, fig. 8), a male, aged $9\frac{1}{12}$ years, presented himself on April 21, 1925, with a swelling, having a history similar to that of Case I, but in the mandibular left deciduous second molar region that tooth was present, carious and with a dead and infected pulp. There was considerable external swelling in this case as the cyst was larger.

The same procedure was adopted as in Case I, a skiagram (exhibit 26, fig. 9) was taken, the deciduous molar removed and the cyst operated on; the piece of tissue removed was saved for microscopic examination. This (exhibit 27, fig. 10) showed a thin, evenly spread epithelial lining to the cyst, two to four cells deep, without epithelial processes; surrounding this was a connective tissue capsule, and external to that, thinned out alveolar bone.

Except for the fact that the cyst was larger and that entire resolution therefore took longer, the subsequent history was very similar to that of Case I, and is illustrated as follows: Exhibit 28 (fig. 11) shows the condition twenty-nine days after operation, merely the tip of the outer premolar cusp being exposed, the tooth now being surrounded and supported by adherent soft tissues. Exhibit 29 (fig. 12), 133 days after operation, shows the tooth erupting well but rather outside the arch. Exhibit 30 (fig. 13), 350 days after operation, shows the tooth now in mal-occlusion with the maxillary second deciduous molar which is locking it buccal to its

FIG. 9.—Skiagram of fig. 8.

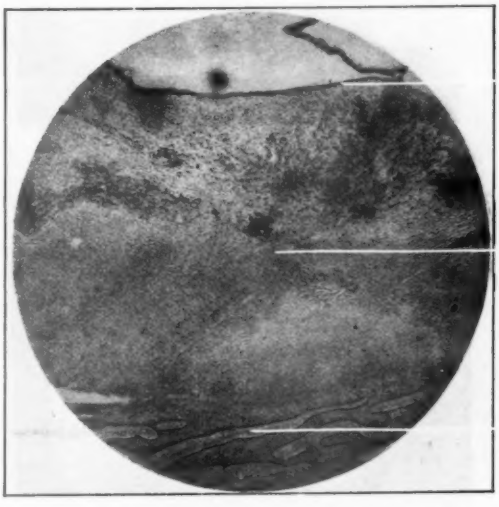


FIG. 10.—Photomicrograph of portion of cyst wall of fig. 8.

FIG. 11.—*Case II*, twenty-nine days after operation.

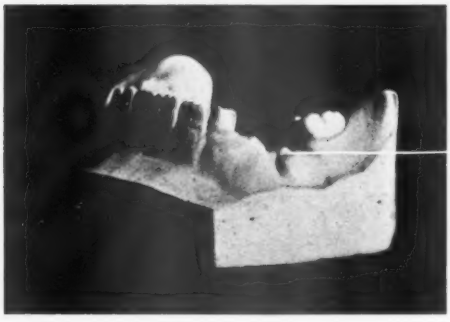


FIG. 12.—*Case II*, 133 days after operation.

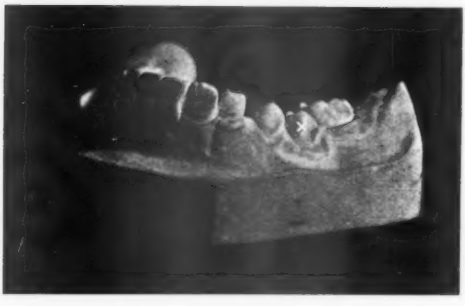


FIG. 13.—*Case II*, 350 days after operation, $\overline{5}$ in malocclusion.

normal position in the arch; the maxillary deciduous molar was ground, and five months later, being a little loose, was removed, and since then (exhibit 31, fig. 14) the premolar which was in the cyst has come into its normal position in the arch and into normal occlusion. The photograph shows the condition 665 days after operation. There has been no mechanical assistance whatever.

Case III.—B.N. (exhibit 32), a female, aged 6, presented herself on December 17, 1925, with a swelling, having a similar history as regards the maxillary right incisor region. Both deciduous incisors on that side were in place, carious and with dead and infected pulps, but on the left side the deciduous first incisor had been lost and the corresponding permanent incisor was about one-third erupted. The skiagram (exhibit 33) shows the right permanent first incisor high up and with an area of rarefaction round its crown; both first incisors are otherwise developed to much the same extent. A piece of tissue, outlined on exhibit 32 and including both deciduous incisors on the right side, was removed and showed (exhibit 34) on microscopical examination with the first incisor *in situ*, a cyst cavity having an epithelial lining of very varying thickness, which, by moving the slide, can be traced almost into direct continuity with the root of the first incisor. Round the apex of this tooth there is much epithelial proliferation, and there are epithelial processes and loops on the outer aspect of the cyst; outside this lies connective tissue and some thinned-out alveolar bone, but in places the alveolar

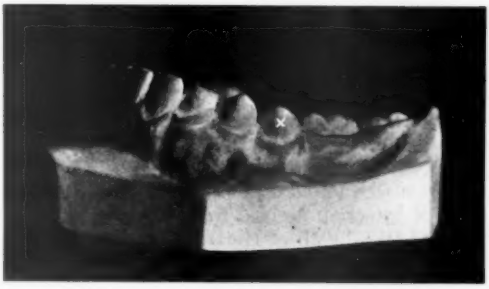


FIG. 14.—*Case II*, 665 days after operation, $\overline{|5}$ in normal occlusion.

bone has disappeared, notably from the area adjacent to the tooth, so that the tooth has no bony socket on its labial aspect; gum, covered by its normal surface epithelium, is seen outside whatever bone is present.

The subsequent history is seen in exhibit 35 which shows the condition eighteen days after operation, with the right permanent first incisor well presenting, and exhibit 36 which shows the condition 102 days after operation, with the tooth erupted still further. I then lost sight of this child for some ten months, but eventually traced her to Devonshire, and through the kindness of a friend obtained the final model (exhibit 37) which shows the condition 397 days after operation, with the permanent right first incisor erupted as far as its fellow of the opposite side, but rather crowded in the arch.

Case IV.—The details of this case need not be given. It occurred in a male, aged 8, in the mandibular right deciduous first molar region, that tooth being present and "dead." The course of the case was similar to that of others. The serial models, skiagram and section are on view as exhibits 38, 39, 40, 41, 42 and 43, and the dates of each are appended.

It will be noticed that in the four cases I have quoted—which are the only cases I have so far treated in this manner—the ages of the patients were very near to the normal eruption dates of the permanent teeth affected by the cysts, and I have

no doubt that this was much in favour of the treatment which was carried out. I have not had the opportunity of trying it in a case at a later age though I should much like to do so. I imagine that the nearer the age of normal eruption the obstruction is removed, the greater and more rapid will be the degree of success attained. But, again, this will depend on the size of the cyst.

In conclusion, I suggest that I have now demonstrated to you :—

(1) That granulomata occur on deciduous teeth.

(2) That dental cysts occur on deciduous teeth.

(3) That when dental cysts occur on deciduous teeth they may envelop adjacent unerupted permanent teeth.

(4) That cysts on deciduous teeth may obstruct, delay or misdirect the eruption of adjacent permanent teeth.

(5) That, on the removal of the obstruction, eruption of the underlying tooth may be resumed.

(6) That, when in the process of its growth a dental cyst envelops an unerupted tooth, a dentigerous cyst is produced,

Of the ten reasons I gave you in support of my thesis in 1922, five (Nos. 1. 2, 4, 9 and 10) were already recognized clinical facts which I incidentally confirmed or demonstrated. Five others (Nos. 3, 5, 6, 7 and 8) I demonstrated at that time, and now again demonstrate in some detail.

You will observe that I have not tried to prove that dentigerous cysts *always* arise from septic, deciduous teeth but only that they very frequently and indeed usually do so.

It has been suggested that a comparison of the different innocent epithelial growths, believed to originate from the epithelial dental formative organs, might usefully be made at this point (exhibit 44, fig. 15).

The *dental cyst* as usually found has been shown by Mr. J. G. Turner to be caused by the stimulus of infection derived from a dead permanent tooth acting on the epithelial remnants of the sheath of Hertwig, that being the only epithelium in the neighbourhood.

The *dentigerous cyst* which involves a permanent tooth I have endeavoured to show as arising in a similar manner, except that the stimulus usually originates in a dead deciduous tooth, but may be derived from a dead permanent molar, when it involves permanent molars. In the case of this cyst, however, there are several groups of cells from which it may grow.

The *cyst of eruption over a deciduous tooth or a permanent tooth which has no predecessor*, though of similar histological structure to both the former cysts, obviously cannot arise from the same cause. It might arise from trauma, but I do not think this is probable unless it is the gentle force of an unusually slowly erupting tooth which is the stimulus. Consideration of this cyst surely emphasizes the important fact that we do not yet comprehend all the possible causes of epithelial cystic growth. In this particular instance there appears to be merely a perversion of an ordinary physiological process.

We may, therefore, surmise that both dental and dentigerous cysts may occasionally be due to such a cause as would give rise to this cyst of eruption, or to some other cause at present unrecognized. An eruption cyst with the deciduous, or any other tooth presenting, is to all intents and purposes a dentigerous cyst, and should consequently, I affirm, be classed as an odontome.

Apart from the effects of infection, of certain light rays, of thermal changes, of trauma and of certain drugs on the proliferation of cells, we know only too little about the causes of cell growth.

Section of Odontology 77

The *cyst of eruption over a permanent tooth which has a deciduous predecessor* may originate either in the same way as the eruption cyst of a deciduous tooth or it may be a cyst caused by a deciduous tooth which has been shed, the cyst remaining in the path of eruption of the permanent tooth, as shown in the diagram.

Finally, with regard to the *multilocular cyst*, we have no certain knowledge about its cause or source of origin, and after the possible sources of origin as shown in the diagram (fig. 15) I would put a large note of interrogation. I recently saw such a cyst present with all three permanent molars *in situ*.

Taking into consideration the recent paper by Drs. Critchley and Ironside on "The Pituitary Adamantinomata," published in *Brain*, 1926 (vol. xlix, part 4), and

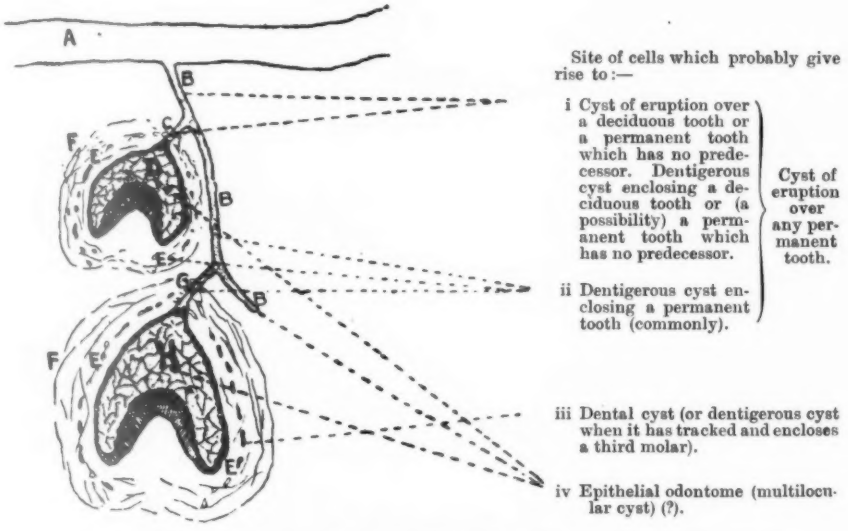


FIG. 15.—Diagram of named parts of epithelial dental formative organs. (This diagram is not true of any age.) A, surface epithelium; B, tooth band; C, remains of neck of deciduous enamel organ; D, deciduous enamel organ; E, sheath of Hertwig; F, tooth follicle or sac; G, remains of neck of permanent enamel organ; H, permanent enamel organ.

referred to in the *British Dental Journal*, March 15, 1927, I think that there may be some similarity in the origin of multilocular cysts and the somewhat similar epithelial growths of the anterior part of the pituitary body, although I am inclined to believe that the histological similarity of the cells of both these growths to those of the normal enamel organ has been somewhat overstated. Apparently the stimulus which gives rise to the multilocular cyst is not necessarily one peculiar to their situation in the jaws such as the immediate vicinity of micro-organisms and toxins to septic teeth or trauma.

It has been thought that the multilocular cyst may be due to an atavistic tendency to polyphyodontism, but one cannot readily see that the intracranial growths can be ascribed to such a cause, although it is true that the epithelium from which

they arise was once in direct continuity with that of the tooth-bearing area. I am reminded of a case reported several years ago in the French press, of the continued backward growth of the maxillary tooth band beyond the third molar, which gave rise to many teeth in excess of the normal number in the neighbourhood of the orbit.

[*Most of the exhibits referred to in this paper have been presented to the Odontological Section of the Museum of the Royal College of Surgeons, England.*]

Section of Ophthalmology.

President—Mr. Ernest Clarke, C.V.O., F.R.C.S.

CASES.

Early Retinitis Pigmentosa.

By Humphrey Neame, F.R.C.S.

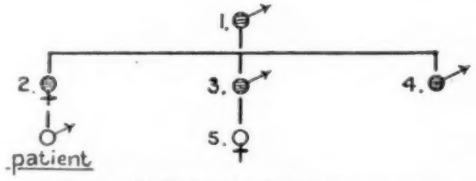
Patient, a male, A. S., aged 24, farm labourer.

History.—Night blindness for two years; has to be led out of doors at night. Complains of spots moving with the eyes.

Vision, right, $\frac{6}{36}$, $\frac{-1{\cdot}25}{-0{\cdot}75-180^{\circ}} = \frac{6}{6}$ at times. Vision, left, $\frac{6}{36}$, $\frac{-1{\cdot}0}{-0{\cdot}5-180^{\circ}} = \frac{6}{6}$ (pt.)

Fine pigment dusting of periphery of fundus.

The family history of the condition covers four generations. Not only the boy's mother, but his maternal grandfather and his great-grandfather were affected. The mother is said to be blind; the grandfather almost blind before he died, and the great-grandfather is said to have had very defective sight.



Explanation of Chart.

Nos. 1 and 2 totally blind. Patient's mother was under Mr. Mayou about 1916 at Central London Ophthalmic Hospital. No. 1 was affected at an early age. No. 5 was almost blind and was under Mr. Mayou at Central London Ophthalmic Hospital in 1914. Both a brother and the father of No. 1 were blind.

Pseudo-pterygium.

By Humphrey Neame, F.R.C.S.

Patient, a male, A. H., aged 69, labourer in cement for forty years.

History.—At age of about 25 had first attack of soreness of eyes; treated by chemist with ointment. He thinks the sight began to fail about three weeks after some cement blew into his eyes, and caused the soreness.

Vision, right $< \frac{6}{60}$; vision, left, $\frac{6}{36}$. Lens clear and fundus normal as far as can be seen. This is a case of encroachment on the cornea. It looks like pterygium; it is conjunctiva-like tissue, and in one place it almost obscures the pupil. In one eye there is an encroachment over three-fourths of the circumference, not only laterally, but below. Probably the condition is due to repeated slight burns of conjunctiva and cornea caused by the cement dust.

66 Goulden: *Membrane in the Anterior Chamber*

Epibulbar Vernal Catarrh.

By CHARLES GOULDEN, O.B.E., F.R.C.S.

THIS is the most severe case of the kind I have ever seen. The patient first attended the hospital on February 28, 1927, and he gave a five years' history of seasonal attacks. This particular attack had lasted since January 13, 1927. A conjunctival swab showed epithelial cells 80 per cent.; no eosinophils; the blood showed 5·5 per cent. of eosinophils. On May 9, 1927, a conjunctival swab showed over 90 per cent. eosinophils, and the blood-count showed 9·5 per cent. eosinophils, while on June 3 it showed 10 per cent. of them.

I have tried general ultra-violet light treatment, but there has been no effect at all. I intend now to try radium locally.

Membrane in the Anterior Chamber.

By CHARLES GOULDEN, O.B.E., F.R.C.S.

THIS man has a membrane in the anterior chamber following a severe kerato-iritis. He is 30 years of age, a clerk, and he first came to hospital in 1911. When 14 years old he attended the out-patient department for 2½ years, but I have no note of what his condition was then. Apparently he was well from that date until 1918, when he attended for six months. He then remained well until August, 1926, when he had pain for several days, also bilateral iritis. He did well, and was discharged on December 5, 1926. I saw him again on January 22 last. In 1914 and 1919 his Wassermann was positive, now it is negative.

The slit-lamp view, which I put on the screen, shows the position of the membrane detached from the posterior surface of the cornea, but adherent to the cornea near its margin. It is a web-like arrangement.

Something like this was originally described by Vogt. The first case of the kind I saw was shown me by Mr. Cardell, but that case differed from this in certain respects.

Discussion.—Mr. HUMPHREY NEAME said that this case showed what he took to be fibrous tissue. In cases of severe interstitial keratitis in which the eyes had to be excised, sections showed a thick and very dense fibrous tissue membrane adherent to the back of the cornea. In the present case the condition seemed to be parallel, except that in this case there had been organization into a layer of fibrous tissue which was largely separated from the back of the cornea.

Mr. LINDSAY REA said that Vogt, in his atlas on the slit lamp, described pittings on the endothelium in cases in which there had previously been interstitial keratitis. He himself had examined a number of these cases, both recent and old, but he had not been able to make up his mind that they were pittings. They seemed to be holes in newly formed connective tissue. He had often seen thickened bands running across the back of the endothelium, but had never seen a case which showed the connective tissue so well as that exhibited by Mr. Goulden.

He thought the explanation of this case was that the connective tissue was formed onthe endothelium, When the eye was in a state of severe keratitis the tension was very low, and it was during this stage that the connective tissue was formed. When the attack had passed over, and the eye had returned to its normal tension, the inelastic connective tissue would not stretch, although the elastic cornea did so, and so the tissue would be pulled away from the cornea.

The scalloped appearance of the upper part of the connective tissue band in this case indicated that the membrane had been stretched through the eye regaining its tension. It was difficult otherwise to understand how connective tissue could be formed in the aqueous.

Section of Ophthalmology 67

Two Cases of True Diabetic Cataract.

By CHARLES GOULDEN, O.B.E., F.R.C.S.

I have called the condition in these two cases true diabetic cataract. Much discussion takes place as to the frequency with which cataract occurs in diabetes; also as to whether we have merely a patient suffering from diabetes who is also suffering from senile cataract, or whether the cataract itself is due to the diabetes.

The condition was originally investigated by Schnyder, who worked out 59 cases of diabetes. Amongst these he was able to find only one case which showed any distinctive features. That case which Schnyder described was, in appearance, exactly like the two cases I have brought to-night. The opacities are very small, white and glistening and some are coloured (such as we associate with the reflection of light from the surface of crystals), immediately under the epithelium of the capsule. In all other conditions an opacity will respect the anterior zone of disjunction which is underneath the anterior capsule but does not invade the area between the zone of disjunction and the anterior band of the lens. But in cases of true diabetic cataract the opacities come up to the under surface of the capsule. If you examine these by oblique light you will see there are many small water-clefts between the most superficial fibres of the lens. The lustre, both from the anterior and the posterior surface of the lens, is little marked, as one would expect when opacities have reached the capsule.

I show you a picture of Schnyder's patient, aged 33. These cases only occur in young people. The following is the description of my cases:—

Case I.—Male, aged 26, 5 ft. 10 in. in height, weight 5 st. 2 lb. There is a $3\frac{3}{4}$ years' history of polyuria and excessive thirst. He was admitted on November, 1923. The sugar was 0·17 per cent.; on diet and insulin it declined to 0·1 per cent. For eighteen months he remained sugar-free. He discontinued insulin for some time, then resumed it. Later he was admitted into another hospital, almost in a state of coma, in November, 1926. He had insulin and dietetic treatment, and was discharged. He was re-admitted in January, 1927. His blood-sugar was then 0·18 per cent., and he is now taking 25 units of insulin twice a day. He is being dieted, receiving carbohydrate 45 grm., protein 67 grm. daily. Blood-sugar 0·125 per cent.

In complicated cataract there are similar opacities, but they invade the axis of the lens.

Case II.—Female, aged 35, who has had diabetes since 1923. She has been in hospital twice, namely, in 1925 and 1926. In July, 1925, it was found that she had signs of cataract. Vision in the right eye was represented by mere shadows, in the left eye it was $\frac{6}{18}$. She had a low renal threshold and glycosuria. She was treated with insulin, and the blood-sugar became normal. Last December I removed the lens from the right eye, and subsequently did a needling. Since then she has been taking 20 units of insulin twice a day. You see fine dots immediately under the epithelium of the capsule, and you will note the ginger appearance on the posterior surface of the lens. The lens shows clearly the lines of separation between the adult and the fœtal nucleus.

Lantern Demonstration of the Filtration Angle.

By Lieut.-Col. H. HERBERT, F.R.C.S., I.M.S.

[PHOTOMICROGRAPHS were shown, mostly from the eyes of old people with more or less senile thickening of hyalin or glass-membrane, to illustrate the following statements.]

The outer trabeculæ or lamellæ of the pectinate ligament become part of the sclerotic simply by losing their hyaline sheaths. A very variable middle division of the ligament passes back to give origin to more or less of the meridional section of the ciliary muscle.

In the inner trabeculæ a change exactly the opposite to the above occurs at the level of the posterior end of the scleral furrow. The white fibrous and elastic centres disappear, while the hyaline sheaths thicken. The bands form an open meshwork, combining maximal

68 Herbert: *Lantern Demonstration of the Filtration Angle*

strength with elasticity, to support the pull of the radial and circular portions of the ciliary muscle. This meshwork, widening posteriorly, ends in a zone of mixed glass-membrane, elastic and collagenous threads, passing backwards and inwards, bordering the angle of the anterior chamber. There is some evidence that this zone results from retraction of radial muscle bundles from glass-membrane sheaths, in which the heads of the muscle were encased. But the innermost bands and threads, before reaching the muscle, are commonly lost in an area of equatorially folded elastic tissue, which has been described as forming a ring close to the base of the iris. And sometimes quite the innermost of the terminal threads, traced from the pectinate ligament, may be said to miss the muscle altogether. They curl round the angle of the chamber to be connected, through the "elastic ring," with the well-known peripheral radiations of the dilator of the pupil, and with the end portion of the external limiting membrane of the ciliary body.

The elastic tissue of the filtration angle is feebly developed in young eyes, and it remains so in eyes in which the ciliary muscle is of the short, bulky, hypermetropic type.

With the backward sloping elongated myopic type of muscle, composed mainly of parallel bundles, the "elastic ring" is found to be stretched out in a line along the sloping border of the muscle, and it is to be noticed that it has only relatively feeble scattered inner connexions with the iris-dilator and with the limitans externa.

There is a senile type of muscle that needs more definite recognition, (*a*) since it presents as great a contrast, in its way, to the myopic type as does the hypermetropic muscle, and (*b*) because of its frequent association with chronic glaucoma. It has three main features: (1) Its inner border projects relatively forward, to give the muscle a cupped anterior surface, the elastic ring lying bunched up in the cup. (2) It has a relatively thick and closely packed inner plate of circular bundles extending back to the beginning of the pars plana, the thickening being at the expense of the middle fasciculi, which are thinned and separated by considerable fibrous tissue. And (3) its connexions with the iris-dilator and the limitans externa, through the elastic ring, are usually strong and defined.

As regards (1), the muscle as a whole does not extend far forward. The sinus of the anterior chamber reaches back distinctly beyond the level of Schlemm's canal, so that the pull of the inner portions of the muscle on the pectinate ligament is backward, rather than inward. There is evidence that this peripheral extension of the sinus is partly acquired.

[An explanation of this acquired extension through the mechanism of accommodation was put forward.]

As regards (2), the formation of the inner plate of muscle, it is seen how the pull of the zonule was transmitted to the muscle by elastic fibres attached to the limitans externa. [Photograph shown.] There is a stretching effect more particularly between the check fibres of the zonule—the orbiculo-ciliary—posteriorly, and those coming from the anterior surface of the lens, anteriorly. And probably the strong attachment of the dilator of the iris (the third feature mentioned above) may aid in producing an accompanying forward growth of the ground-plate of the ciliary body, adherent to the base of the iris, that is found in these eyes. This strong attachment to the dilator of the iris may be compensatory to the relatively weak attachment of the inner muscle to the pectinate ligament.

The reality of progressive changes to produce the two opposing types of muscle—this senile type and the myopic—are well illustrated by two photographs from one eye. Above the cornea there is a muscle with a typically cupped anterior surface, in which the elastic tissue lies: while below the cornea, under the influence of cyclitic membrane, the inner muscle bundles have rolled back so completely that the myopic type is reproduced. The elastic tissue lies stretched out in a line along the backward sloping margin of the muscle.

A peripheral extension of the sinus of the chamber, distinctly beyond the possible normal limits, is a feature of some early chronic glaucomas, evidently a result of plus tension in the chamber, accentuating the previous feebleness of the opening pull of the ciliary muscle on the pectinate ligament, which is to be regarded as the primary cause of the glaucoma.

Discussion.—Mr. THOMSON HENDERSON said that Colonel Herbert had given a mass of detail, beautifully illustrated, but he (the speaker) did not altogether agree with him. The unfortunate thing in the descriptive anatomy of the angle of the anterior chamber was that the earlier anatomists described and noted the salient features of the angle only in the lower animals, while later observers had mistakenly applied the same terms to the human eye. The result was a mass of practically meaningless details without guiding principles. Twenty years ago he (the speaker) read before the Ophthalmological Society a paper on the anatomy of the so-called ligamentum pectinatum iridis,[1] and to-day he was able to confirm everything he said then. He could now go further and say that there was no such structure as the pectinate ligament in the human eye. The pectinate ligament of the lower animals was lost in the process of evolution, and the determining factor was the evolution of the mechanism of accommodation.[2] As soon as it was realized that this latter was the determining factor in the configuration of the angle of the anterior chamber in mammals, then one was able to appreciate that the arrangement of the anatomical structures followed in orderly sequence.

Mr. E. TREACHER COLLINS said that in 1899, at the International Ophthalmological Congress at Utrecht, he demonstrated the development of the pectinate ligament and the changes which had occurred in it during its process of evolution, much in the same way as Mr. Thomson Henderson had just done. He (the speaker) pointed out that in most of the mammalia the angle of the chamber was not prolonged out so far as in the human eye. In the latter it extended a considerable distance beyond the canal of Schlemm, in the ungulata it terminated on a level with it. In mammals he described the pectinate ligament as consisting of pillars of the iris, the fibrous strands which Mr. Henderson had shown at the angle of the chamber, a cavernous portion, and then the laminated portion, which Mr. Thomson Henderson termed the cribriform ligament.

In comparing the different forms of pectinate ligament in mammals with human fœtal eyes, it was seen that in the latter it passed through, in its development, stages similar to those found in the mammalian eye. In the human fœtal eye could be seen a cavernous zone, and even fibrous pillars of the iris. He had associated the changes in the pectinate ligament which had taken place in mammals and in the human fœtus with the alteration in the size of the cornea; and he had given measurements of the cornea in relation with the rest of the globe, showing that as it became reduced in size the angle of the chamber was prolonged outwards.

A most important practical point was that in congenitally microphthalmic eyes and in some buphthalmic eyes, the pre-human or pre-natal condition of pectinate ligament persisted, and tended to produce obstruction at the angle of the anterior chamber which, in some cases, resulted in congenital glaucoma.

Colonel HERBERT (in reply) said that he did not mind what the tissue was called. For the human eye he thought "cribriform" ligament was a better term than "pectinate." But he accepted the terminology commonly used in this country, though not in Germany. What Mr. Henderson called the pectinate ligament in the horse, for instance, was known in Germany as the uveal meshwork of the iris angle, and it was composed of uveal tissue.

[1] *Trans. Ophthal. Soc. U.K.*, 1908, xxviii, 47.
Ibid., 1926 xlvi, 280.

70 Herbert: *Lantern Demonstration of the Filtration Angle*

The criterion was the presence of pigment cells. Mr. Henderson's "cribriform ligament" was a different structure; it was the scleral meshwork of the iris angle. Its strands consisted partly of hyaline membrane, and there was also some elastic tissue. In human infants there was often a good deal of uveal meshwork remaining, but it did not persist.

He had confined himself to matters of practical bearing. He did not claim to have proved anything by the photographs beyond isolated facts; each observer must form his own conclusions.

Section of Orthopædics.

President—Dr. W. T. Gordon Pugh.

The Treatment of Spastic Paralysis by Sympathetic Ramisection.

By Norman D. Royle, M.B., Ch.M. (Sydney University).

Mr. Royle said it was not his intention to read a paper; he assumed that members of the Section had already read most of the literature on the subject. His intention was to show cinematograph films of patients at different stages which had been taken for the purpose of illustrating the changes brought about by the operation of sympathetic ramisection. He had, of course, read the criticisms which had emanated from the British Isles and from the United States of America. He had just passed through the latter country, and had found that there the operation was universally accepted—i.e., more or less so—and even in Boston he had found Dr. Harvey Cushing in favour of it. The cases in which he (the speaker) performed the operation in Boston, U.S.A., were not failures, but he had found results there which were not perfect, as they left the children with contractures.

He would first refer to the effects seen in animals after cutting sympathetic nerves. The immediate effect was to cause vaso-dilatation. This became evident in a thin subject as soon as the nerves had been cut; in a fat subject the change was delayed for an hour or two.

The slide illustrating this change was given to him by Dr. Brown, of the Mayo Clinic, which he (the speaker) had recently visited. The case was one of spastic paraplegia. There was an increase of surface temperature following this section, and in one case, that of Dr. Cushing (Boston), in which he (the speaker) had performed the operation, the change was 7° F.

The next feature was the change in muscle-tone. He took, for this purpose, the definition of muscle-tone given by Holmes and Walshe in Nelson's "System of Medicine," and he would demonstrate by the slide the way in which muscle-tone was manifested. In a tonic muscle the reflexes were rapid and jerky. When a muscle was hypotonic, the relaxation was slower, so that the leg, after eliciting the knee-jerk, fell more slowly and gradually to the resting posture.

He would next demonstrate by a cinematograph film the behaviour of the limbs after sympathetic ramisection: also, in a goat, after decerebration. The limbs in which the sympathetic nerves had been divided behaved differently from those in which

64 Royle: *Treatment of Spastic Paralysis by Sympathetic Ramisection*

these nerves were intact. Criticism had been levelled at these experiments by those who tried them on the cat, but the cat showed a tendency to curl up, and for that reason the goat was a better animal to study. He himself used goats. The limbs also showed a different behaviour when the animal was anæsthetized. One could remove the sympathetic nerves from either the fore- or the hind-limbs of the animal, and one would always see a collapse of the limbs from which the sympathetic nerves had been removed.

In a decerebrate animal one could see the shortening reactions in the quadriceps muscle. This result was consistent. He wished the rate at which the limb fell and the final posture of the limb to be particularly noted. He showed the reaction in an animal 120 days after decerebration. In the human subject the knee-jerks were very brisk, and then there was a slow subsidence to the original position of the limb. But the passive posture was not so well maintained. After sympathetic ramisection the subsidence was much more rapid.

In the case from which the next film was shown a very considerable change took place. Before this operation the limb behaved as shown in the film, whereas after the operation it comported itself as if it possessed no tone at all. This patient gave him a very interesting piece of information. He asked this man what change he experienced in his lower limb as a result of his operation, and he replied that before the operation he was unable to relax, i.e., if his leg went out into the position of extension, there was no movement in it except a reflex one; whereas after operation he gained fair movement in the first week, and he could now relax.

That brought one to a consideration as to how muscle-tone was normally controlled. In ordinary circumstances we had no control over muscle-tone, except as a concomitant of active movement. We could close our eye and open it, but when it was once open it remained more or less in that position without intervention; we could not render it more fixed or less fixed.

In lesions of the central nervous system, two things might happen: the patient might lose control over his phasic movement; secondly, he might lose ability to let go his muscles. In addition, there was a release of function in the centres controlling movement and in those which controlled tone. If, under ordinary circumstances, we had no control over tone, then, under these abnormal circumstances, tone became an obstacle against which the patient had to move. This tone affected all groups of muscles, not only extensors, but, in spastic paralysis, the flexors also.

At the end of twenty days after the operation this particular patient had the amount of movement shown on the film. Before the operation he could not control movement at all.

In the next case the patient had gained a certain range of movement after the operation, but flexion was not very good, as both limbs were involved and the muscles of the trunk also, and there had been no effective after-treatment. He went away to Queensland after the operation, and the speaker saw him a little over a year afterwards. He still retained the characteristic reflexes depicted in the film.

In examining these patients it was important to distinguish between clonus and lengthening-and-shortening reactions. In the next series this was well shown. Before operation there was a shortening reaction in the quadriceps, and after the operation clonus appeared. The first of these patients was one whom he had observed for many years.

Previously she had undergone Stöffel's operation for the adductors and flexors of the knee, and for the peroneal group of muscles, but this did not enable her to balance. The result seen, a month after ramisection, was better than in the years preceding it, though before the last operation, education of muscles had been carefully attempted. The condition in her case was congenital. Twenty-eight days after operation the result was even more satisfactory.

In the next case shown, the right upper limb had been so useless that the patient could not learn to distinguish one sensation from another. On the day following ramisection, however, the man was able to close his hand over the bandage.

The next patient, a boy, had adductor spasm, and, in addition to performing ramisection the speaker lengthened the hamstring muscles. Twelve months after the ramisection the patient was able, as the film showed, to walk without aid other than that of a stick.

The next patient, a woman, aged 23, was the subject of congenital spastic paraplegia. The foot extended and dorsiflexed in co-ordination with the knee. There was no delay in relaxation; the knee-jerk was good and brisk. Six weeks after bilateral sympathetic ramisection she was able to walk quite passably with the help of two sticks, though she had never walked previously in her life.

The subsequent history of the foregoing patient was interesting because of the work which had been done by the Russian school. It had been shown by Russian investigators that removal of the sympathetic conferred upon these patients the ability to resist greater and greater amounts of fatigue. This patient, after the operation, was able to walk only a few yards, but subsequent improvement in this respect was so marked that she was later able to walk miles—as stated in a letter he had received from her. These patients could walk because there had been taken away something which enabled inhibition to be practised. This patient could now balance herself without support.

Another patient showed, after the operation, a change in the range and speed of active movement. She could not inhibit the quadriceps muscle when told to let the leg drop. Another feature to note in her case was the manner in which she carried the limb forward before operation, in comparison with what she could do after it. She had previously undergone, in this country, operations which were chiefly tenotomies. Six months after sympathetic ramisection she was able to walk moderately well without a stick or other support, and there was a satisfactory alteration for the better in the range and speed of her movements.

The next was a patient who, also before ramisection, was unable to balance on the limb. He was one of the patients in whom the spastic element had more or less disappeared and left him with contractures. When contractures were present, his (the speaker's) plan was to do sympathetic ramisection on one side, treating also, on one side, the hamstring muscles, tendo Achillis, and whatever else was necessary. This he did at one sitting. After operation the patient was able to walk up and down stairs fairly well; he could also run. He was shown walking without boots.

This patient also illustrated a change which was produced by, or following, the operation in regard to the bowel condition. Most of these patients, before operation, had more or less obstinate constipation. At operation this man was noticed to have a dilated bowel, though no skiagrams of it had been brought. After ramisection had been performed, he was able to have a natural daily motion without any aperient at all.

The latest application of this operation, so far as he (Mr. Royle) was aware, was to Hirschsprung's disease, a case of which was operated upon by Dr. Wade, of Sydney.

The patient was a boy, aged 10. He had never had a normal motion, he had always to have an enema or an aperient for the purpose; ever since operation he had had a normal motion every day.

66 Royle: *Treatment of Spastic Paralysis by Sympathetic Ramisection*

For this condition the operation differed from that in the ordinary paralytic case, as the rami which were divided were those going to the inferior mesenteric ganglion; and the operation was done on one side only. In the United States, especially at the Mayo Clinic, he was told that no effect had resulted from the operation, but he (the speaker) considered that that was because the surgeon did not go high enough. In operations of the kind done on the goat the number of motions subsequently passed was definitely increased.

The next patient shown was a boy, aged 16, who had never walked. He had undergone tenotomies, but no other treatment. His limb was more or less flexed at the knee. Seen in the act of walking for the first time, the limb was still flexed. This was the boy whose case was commented upon by the *British Medical Journal*. At the end of six months after the operation the boy was able to play tennis, and to bowl fairly well in a game of cricket. The film showed him playing the former game. His was a case of congenital spastic paraplegia. Immediately before the operation his range of movement could be described as *nil*. The film showed his first attempt at standing, also his first essay at walking. He was later able to walk with fair rapidity, but with obvious difficulty. Three months later his walking attempts were much better. He had hallux valgus, which at this stage had not been corrected. Before the operation his knee-jerk could not be obtained; the limb was so rigid that it remained in any position in which it had been placed, even for half an hour at a time.

The next film showed the increased range of active movement three months after operation. At the end of six months he was playing tennis.

When he (Mr. Royle) first introduced this operation for spastic paralysis, he thought that spastic chorea and athetosis were contra-indications for ramisection, but that was because he had not tried it for those conditions.

The first of the patients he was now showing was a little boy whom he had observed for a number of years, and who later went to live in one of the other Australian capitals. He came back to the speaker three or four years later. He then had some degree of spasticity in his hands. The outstanding feature of the case was the presence of choreiform movement.

A girl aged 19 had suffered from spastic chorea all her life and was unable to sit unsupported. She expressed her willingness to try anything which seemed likely to benefit her. One heard a great deal about the "cruelty" of conducting experiments on animals, but often human beings who were suffering showed a good deal of courage in inviting operation which might be in the nature of experiment. In these cases the operation meant conferring on these patients the power of inhibiting in some degree these involuntary movements. The effect of the operation was well seen on the film. In this case the choreiform movements affected the upper limb, and also the facial muscles to some extent. Four weeks after sympathetic remisection the jerky movements had not quite gone, though little remained. The patient could open and close the hand fairly well.

The next patient was aged 19, and affected with diplegia and chorea. It must be remembered that the film was taken under exceptional circumstances; in public, and with the patient hypersensitive on account of the affliction. Before the operation she could not walk more than a few yards, but after it she could walk some hundreds of yards, and was able to sit unsupported. Part of this patient's disability was due to Stöffel's operation having been performed. He (the speaker) had previously removed a section of the nerve supply to the calf, and that deprived her of some support which she would otherwise have had.

The next patient, a boy, had previously had tenotomies, and arthrodesis of the ankle. The next part of the film was taken as soon as the patient was out of bed, and the later part was taken a week later. The second part of the film was by slow motion. He had stiff ankles, but was walking without support.

The next exhibit was shown as an answer to those who said the results attained were due to education, or re-education of muscles.

This child was 18 months old, and before operation was unable to use its hands; any opening of the hand had to be carried out passively. The film showed the same child seven days and four weeks after operation respectively. At the latter date it was able to reach out to and grasp articles.

The next film was to show the result obtained in post-encephalitic rigidity. He had prepared films of two such patients.

The history and condition in the first was, from the neurologist's standpoint, as follows: There was no history of illness or accident previous to the onset of the present condition. He was quite well until a fortnight before Christmas, 1923. While at work one day he felt ill and weak and had to go home. He was in the hospital three days and then became unconscious and delirious. The delirium lasted four days. The patient was able to leave the hospital a fortnight later, but all his limbs felt weak, the left upper and lower extremities being most affected. At this time, too, he became obstinately constipated. This condition had, at the date of the note, grown gradually worse and the facial expression had become more and more fixed. Speech was slow and monotonous. There was, apparently, no disturbance of sensation in the head, trunk, or extremities.

With regard to the motor system, the left upper extremity was held fixed and was adducted across the chest. This arm did not swing when the patient walked. Objectively, there appeared to be rigidity of muscles of the left leg. The patient complained of generalized stiffness and weakness of movements. In all the extremities the movements were performed slowly, more so on the left side than on the right, though there was no real paralysis, nor were there any tremors. The knee-jerks were exaggerated, the plantar reflex was flexor. There was no clonus. The abdominal and cremasteric reflexes were active. The pupils were equal, but reacted somewhat sluggishly to light and accommodation. Tests for syphilis, applied to blood and cerebro-spinal fluid, were negative. While in bed, the poverty and difficulty of movements were not obvious, but when the patient was up it was seen that he had considerable motor disability. There was an attitude of generalized flexion, the gait was slow, unsteady and laboured, sometimes tripping and festinating. At times this patient became so rigid that he was immobilized. Injections of hyoscine relieved the muscular rigidity within about four hours, the effect gradually wearing off in two or three days. Injections of saline solution were given, the object being to exclude the factor of suggestion, but these did not influence the rigidity. While under observation the rigidity was apparently slowly progressive.

The patient was re-examined in January, 1927. A striking change was then seen as compared with the condition before operation. He now sat erect with both upper extremities in a normal attitude, i.e., resting on his thighs. Previously the left upper extremity was flexed across the chest. The patient's facies was now much more mobile and lively, but expressions still tended to fade slowly. Movements of both upper extremities were now comparatively free at the proximal joints. The finer movements of the fingers—the piano-playing movements—were poorly performed, but movements of flexion and extension were now better than before. The patient's gait was active and lively, and he ascended and descended stairs without difficulty. Before the operation the patient could neither feed himself nor dress himself, but he now did both without difficulty. His bowels were now evacuated daily and without the aid of aperients; previously he had been constipated.

Another case was that of a patient whose left lower limb was rigid before operation. After the operation he walked fairly well, but took very short steps. He could now also run. Dr. Pope had performed the operation.

In conclusion, he (Mr. Royle) said he had shown these pictures in order to prove that something occurred after division of the sympathetic nerves. His contention was that the sympathetic system controlled tone, and that the operation removed the hypertonia, conferring on the patient the ability to move more easily and through a greater range, and to ensure a better balance. There were also other subsidiary effects, but he would leave the matter there.

68 Royle: *Treatment of Spastic Paralysis by Sympathetic Ramisection*

Dr. W. T. GORDON PUGH (President) said that of all the diseases which members of the Section were called upon to treat in children, the most disappointing, with regard to results, was spastic paralysis. Therefore it was of great interest to all the Section when it was announced that Mr. Royle and Dr. Hunter had invented and perfected a satisfactory method of treatment. As Members were aware, Dr. Hunter had died, after an extraordinarily brilliant career, at the early age of 26.

He thanked Mr. Royle on behalf of the Section for his lucid and interesting exposition of the subject.

Section of Urology.

President—Mr. W. Girling Ball, F.R.C.S.

Clinical and Pathological Meeting.

Villous Tumour of the Pelvis and Ureter.

By J. Swift Joly, F.R.C.S.

C. P. L., male, aged 71. Noticed urine was discoloured off and on during May and June, 1926. Early in August profuse hæmaturia. Seen by a surgeon, who found that the prostate was enlarged and the bladder filled with blood-clot. Bladder evacuated, and catheter tied in. Hæmorrhage ceased in four days. Catheter removed and patient returned home. Seen by me November 2, 1926. Had several attacks of hæmaturia since August. Complained of pain in back and both loins. Left kidney enormously enlarged, hard, irregular, but freely movable. It filled up the whole loin, and extended almost to the middle line. Right kidney not felt. Prostate: Large adenoma. X-ray examination: Enormous left renal shadow filling up the whole loin, and encroaching on the shadow of the vertebræ. Right side, renal shadow normal in size and position, numerous shadows below its lower pole (abdominal glands). Thorax: No evidence of metastases. Cystoscopy: Neither ureteric orifice visible on account of large intravesical projection of prostate.

Operation, November 10, 1926.—Kidney removed enclosed in its fatty capsule. Ureter thickened in its upper portion, divided where it appeared to be normal.

On section: the kidney was greatly dilated, and contained about 30 oz. of deeply blood-stained fluid. Villous tumour chiefly in the lower calyx, but extending into the pelvis. Secondary growth in ureter. Large hæmatoma in perinephritic tissue, at level of lower third of kidney. This gave the appearance of a solid tumour when the kidney was palpated before operation. Microscopically: Benign villous growth. No evidence of neoplasm in hæmatoma.

After-history.—Convalescence uneventful, but patient still complained of pain in the back. Seen again on December 29, 1926. Had steadily lost ground since operation. Liver enlarged and hard, projecting 2 in. below costal margin. Angular curvature of spine in upper lumbar region. Reflexes normal, no sensory changes. Patient died a fortnight later. No post-mortem examination allowed. No clinical evidence of any other neoplasm.

Villous Tumour of the Middle Calyx.

By J. Swift Joly, F.R.C.S.

H. B., boy, aged 13. Intermittent hæmaturia, first noticed in October, 1926. At first slight, but gradually becoming more severe. Seen by a surgeon early in December. X-ray examination negative. Cystoscopy: Normal bladder and ureters. Clear efflux both sides. Urine from both kidneys and bladder inoculated into guinea-pigs with negative result. Seen by me January 25, 1927. Passing bright red blood in urine, and complaining of a slight amount of pain in the right side. X-ray examination: Outline of right kidney larger than that of left. Cystoscopy: Right renal hæmaturia, clear urine from left kidney. Pyelogram (right side) dilatation of upper and lower calyces, shadow of renal pelvis and middle calyx partially obscured. Left ureter catheterized; urine from this side was normal, and gave urea concentration of 3·4 per cent. after a urea meal of 15 gr.

48 Swift Joly—MacDonald—Neligan—Perrin

Operation, January 31, 1927.—Right kidney exposed, slightly larger than normal. Pelvis filled with a soft solid mass. Nephrectomy.

On section: Soft villous tumour found filling the whole of the middle calyx. Renal pelvis filled with a mass of blood-clot, which had become moulded into its shape, and was adherent to the growth. Kidney substance well preserved, but stained in places by blood. Microscopic examination: Benign villous tumour. In the stained areas of the cortex, the uriniferous tubules were filled with blood.

Convalescence uneventful.

Specimen of Primary Carcinoma of Ureter.

Shown by SYDNEY G. MACDONALD, F.R.C.S.

SPECIMEN removed from a female aged 69. Microscopically it is a spheroidal-celled carcinoma. Patient had had hæmaturia for one month. Cystoscopy revealed a large open ureteric orifice on the left side, and in the distance the lower portion of the growth was seen. The lower 4 or 5 in. of ureter was removed, along with the ureteric orifice and surrounding part of bladder wall . . . the lower end of ureter remaining being transplanted into the fundus of bladder.

Patient made an uneventful recovery.

Congenital Hydronephrosis.

By G. E. NELIGAN, M.B.

PATIENT, a male infant, aged 8 months.

History, two days.—Spasms of abdominal pain. Frequent vomiting. Bowels open at onset, but absolutely constipated since.

On admission.—Temperature 102° F., pulse 140. Child looked very ill. Abdomen greatly distended. Tender lump in right iliac fossa. Difficult to feel distinctly as child was so rigid and distended.

Operation.—Right paramedian incision. Intestines distended. Lump found to be large right hydronephrosis full of purulent urine. Left kidney felt normal. Right nephrectomy.

After-history.—Child was very ill for two days, then made an uninterrupted recovery.

Ectopic Kidney with Triple Ureter, removed from a Man, aged 41.

Shown by W. S. PERRIN, M.Ch., F.R.C.S.

History.—Three attacks of pain in left lumbar region radiating to pelvis and associated with hæmaturia and frequency. First attack, four months before admission. Second attack, two months before admission. Third attack, four days before admission.

Cystoscopy on day of admission. Blood seen passing from left ureteric orifice.

Pyelography showed moderate hydronephrosis of a kidney lying in the hollow of the sacrum and a normal right kidney in usual position.

Bimanual examination showed a tender swelling in pelvis.

Urine: Specific gravity 1010, one-eighth volume of albumin. Blood-cultures showed streptococci but no tubercle bacilli.

Renal efficiency test: Blood-urea 0·06.

The kidney was lying in the hollow of the sacrum. It possessed three ureters uniting to form a small sac which opened into the bladder by a single orifice. The sac contained a large calculus which obstructed the upper two ureters entering it.

Normally Placed Right Kidney possessing Two Pelves and Two Ureters Opening Separately into the Bladder, the Centre Part of the Kidney between the Pelves being Occupied by a Grawitz Tumour.

Shown by W. S. PERRIN, F.R.C.S.

THE specimen was removed from a man, aged 54.

History.—Recurrent painless hæmaturia for eight months, with occasional passage of clots which caused difficulty of micturition.

Cystoscopy showed two ureteric orifices on the right side of the bladder. Blood was passing from the upper of the two orifices. Indigo-carmine from left ureter in ten minutes, and from right lower ureter in ten minutes, none observed passing from right upper orifice in twelve minutes. Blood-urea : 0·058 per cent. Urine sterile. Nothing abnormal except a few red cells.

Pyelogram showed two pelves to the kidney on the right side, the lower rather larger than the upper.

Specimen of Closed Tuberculosis of Kidney.

Shown by C. E. SHATTOCK, M.S.

PATIENT, a female, aged 35. Left hip had been diseased since the age of two years. She had had thirteen operations on it. Attack of pleurisy last year.

Attended hospital for pain over left hip and was X-rayed.

X-ray examination showed calcified left kidney and ureter, and healed tuberculous hip. Left ureter could be felt on abdominal palpation.

Patient has had no urinary symptoms. Urine normal on examination.

Cystoscopy showed normal bladder.

Indigo-carmine excreted in good time by right kidney, no excretion by left.

Operation.—Calcified kidney and ureter removed; only a short piece of ureter at the lower end was normal. Good recovery.

Specimens from Three Cases of Stricture of the Ureter.

Shown by H. P. WINSBURY WHITE, F.R.C.S.

THE first is a post-mortem specimen removed from a man, aged 56, who died as a result of a head injury. It consists of the kidneys with the ureters attached. Each ureter is the seat of an extensive fibrous thickening. That of the right side situated at the level of the lower pole of the kidney is 1½ in. long and is giving rise to a thickening of each opposite wall of the ureter of 3 mm. The stricture of the left ureter is more extensive, occupying the middle third, 2½ in. in length, while the opposite walls vary from 1½ to 3 mm. in thickness.

In neither case has the fibrous zone contracted to such an extent as to cause any marked narrowing of the lumen of the ureter, with the result that there is only early dilatation of the kidneys. There was a twenty-five years' history of renal colic and no record of any stone having been passed.

50 Wildbolz: *The Indication and Execution of Prostatectomy*

The second specimen consists of the left kidney with the upper third of the ureter attached which I removed by operation from a man, aged 26. The lowest ¾ in. of the ureter shows a diameter of 6 mm. This is in marked contrast with the whole of the remainder of the ureter, for above that point it is only 2 mm. in diameter, and is moreover the seat of four areas of slight narrowing situated equidistant from each other. The result of this constriction is clearly shown in the pelvis, which is the seat of an early dilatation and some inflammatory change.

The patient had a two years' history of pain in the left side, which disappeared following the nephrectomy.

The third specimen I removed by operation from a girl, aged 4½. It consists of a left kidney with two ureters. The kidney and ureters are all dilated.

At operation the two ureters were divided just outside the bladder, and an inspection of the divided ends shows an absence of dilatation in one while there is but a slight distension of the other in this region, indicating that the actual obstruction is in the bladder-wall or at the ureteric orifice.

The first signs of disease followed an attack of bronchitis one month before operation. The medical practitioner who brought the case to me stated that pyuria, dysuria, and some frequency of micturition, together with tenderness in the left renal region, were subsequently noted. On examination, before cystoscopy, the urine was found to be loaded with pus, but there were no localizing signs as to its origin. Cystoscopy was difficult on account of the gross infection of the bladder, and at the second attempt, although no ureteric orifice could be seen, the fluid medium in the bladder suddenly clouded, following pressure on the left kidney.

The kidney and the whole extent of both ureters down to the bladder-wall were removed through two separate incisions. The patient made a good recovery and the urine ultimately became quite free from pus cells.

[*May* 26, 1927.

The Indication and Execution of Prostatectomy.

By Professor HANS WILDBOLZ (Berne).

IT may seem perhaps a little out of place to speak on prostatectomy in England, where Freyer popularized the operation. My reason for doing so is because I thought you might like to hear some foreign ideas on the subject. I therefore hope it may be of interest to you to hear about the perineal operation, which is so seldom employed in your country.

Before explaining the technique of perineal prostatectomy I wish to say a few words as to the indications for the operation in general, in cases of benign hypertrophy of the gland.

The general indications admitted by all surgeons are:—

(1) A permanent retention of a considerable quantity of urine in the bladder (from 150 to 200 c.c. or more).
(2) Frequent attacks of complete retention.
(3) Long-standing infection of the bladder.
(4) Severe repeated hæmorrhage from the hypertrophied prostate.

I know that many surgeons are extending the indications for prostatectomy still further. They think the operation is indicated for patients suffering from frequent micturition, even when they have only a slight degree of urinary retention. They think it best to operate on patients in the early stages of disease before there is any

risk to life. I do not consider it advisable to perform prostatectomy as a prophylactic operation. It is never a harmless intervention, and I think it only safe to do the operation when the patient is in real danger from the disease. But I do not wish to enter into details as to the indications in early cases; I prefer to consider the question: When is the operation safe?

In the early days, a patient with chronic retention of urine was submitted to operation at once. The mortality was very high, the reason being that owing to impairment of the renal function many deaths occurred from uræmia. Later it was admitted by all that one must not perform the operation until the patient's renal function had been investigated. Even now we are not all of the same opinion as to the best tests of renal function, and as to when this function is sufficient to permit the performance of prostatectomy without exposing the patient to undue risk.

I have tested the renal function in each of 135 patients before operation by the following methods:—

(1) By testing the power of the kidney to dilute and concentrate the urine (water test).
(2) By the phenolsulphonephthalein test.
(3) Estimation of the blood-urea.

These examinations were performed several times in each patient, the reason being that usually the results of the tests were so bad when the patient was first seen that operation could not be risked, and the patients had to be treated by regular catheterization for several weeks before it could be performed. One of these patients died after the operation from uræmia, and another, who died from pneumonia and acute bilateral pyelonephritis, showed no signs of uræmia for the first three weeks after operation, but a few days before death the blood-urea rose to 220 mgm. None of the other patients showed any sign of uræmia after operation in spite of the fact that many had shown some deficiency of the renal function when examined.

(1) *The water test* is considered by many surgeons to be the most important of all the tests for renal function. Suter, of Basle, refuses to operate unless the patient can concentrate his urine to a specific gravity of at least 1017. Lehmann considers 1018 and Rubritius 1015 to be the minimum. My figures show that these figures are unnecessarily high, as in twenty-six cases the patients recovered in spite of a much lower concentration of the urine. In eighteen it was from 1011 to 1014; in seven it was 1010, and in one, 1009.

In judging the renal efficiency some surgeons lay more stress on the difference between the highest and lowest figures for the specific gravity obtained with the test. Craig Gordon considers the prognosis bad if the specific gravity of the urine does not vary at least 15 degrees. Salomon, Duttmann and others consider a difference of 10 degrees sufficient. Seventeen of my patients recovered after the operation, although they showed a much smaller variation in the specific gravity of the urine. Several of them showed a difference of only 4 to 5 degrees; two had a difference of only three degrees.

A bad response to this test is not an absolute contra-indication to prostatectomy; it is only an indication that the renal function is impaired. On the other hand, a good response supplies valuable proof of a good renal function. I have never seen a bad result of the phenolsulphonephthalein test when that of the water test was good, and only in two cases where the latter test was satisfactory was the blood-urea abnormally high.

(2) *Phenolsulphonephthalein Test.*—A good response to the phenolsulphonephthalein test shows a satisfactory renal function, but, on the other hand, a bad response does not easily give a definite contra-indication to operation. Negro and Colombet, of Marion's Clinic, state that one should not operate if the patient does not eliminate at least 42 per cent. of phenolsulphonephthalein in the first hour.

Blanc considers 55 per cent. as a minimum, but if I accepted these figures I should have been compelled to refuse operation to many of my patients. The majority of them eliminated more than 30 per cent. of phenolsulphonephthalein in the first hour, but thirteen of them only eliminated between 20 to 30 per cent., and ten less than 20 per cent. But I must admit that most of the patients who showed a low elimination during the first hour had remarkably good elimination during the second, and the total amount of the drug eliminated during the two hours was satisfactory. My conclusions are that an elimination of less than 10 per cent. during the first hour is a contra-indication to prostatectomy. With an elimination of from 10 to 20 per cent., operation is allowable, provided the elimination is as high or even higher during the second hour.

(3) *Estimation of the Blood-urea.*—These two tests should be supplemented by a determination of the urea of the blood, as, if they are satisfactory, the operation is permissible, but if they are not satisfactory they leave us uncertain as to whether it is possible to operate or not.

The estimation of the blood-urea shows us when operation is definitely contra-indicated, but leaves us uncertain as to when it is permissible. A high blood-urea is a contra-indication to prostatectomy, but the normal amount of urea in the blood is no proof of satisfactory kidney function.

Judin, Negro, Colombet, Young and others have seen patients with a normal blood-urea presenting symptoms of uræmia after prostatectomy. I have observed several patients with from 30 to 40 mgm. of urea per 100 c.c. of blood eliminating the phenolsulphonephthalein very badly, and with such a small power of concentration that prostatectomy appeared to be too dangerous. So I believe that the estimation of the blood-urea only indicates a serious deficiency of the renal function later than the water or phenolsulphonephthalein test.

On the other hand, an abnormally high quantity of urea in the blood is certainly a proof that the renal function is for the time too poor to allow of operation. The observations of Cohen, Dodds and Webb, of Grauhan, Gulecke, Salomon, Judin, Illyes, and also my own, show that prostatectomy will certainly be followed by uræmia if the urea of the blood is more than 100 mgm. in 100 c.c., and probably so if it is over 80 mgm. per 100 c.c. When the blood contains from 50 to 80 mgm. the operation is always dangerous, and should only be performed if the other tests of the renal function give good results. These observations show us that the indications for operation should not be based on a single test of the renal function, but it is only by a combination of several tests that we are able to judge accurately of the capacity of the patient's kidneys.

It is obvious from a large number of cases that the three methods give similar results. The estimation of the renal function is then easy, but occasionally the result of one of the tests may appear to be in contradiction to that of the others. For example, if the blood contains more than 80 mgm. of urea per 100 c.c., prostatectomy should not be performed, even if the other tests give good results. If one of the tests of elimination gives a poor result and the other is satisfactory, prostatectomy may be performed if the blood-urea is low.

The only death from uræmia I have had after prostatectomy was in a case in which I operated in spite of unsatisfactory renal function tests. The patient had complete retention and was treated by regular catheterization for three months. He showed very poor power of concentration and dilution of urine, and had 85 mgm. of urea per 100 c.c. of blood, and eliminated 15 per cent. of phenolsulphonephthalein during the first hour and 8 per cent. during the second. The patient's health was remarkably improved by catheterization, and he insisted on the operation. I removed the prostate under sacral anæsthesia, and there was no serious bleeding and no infection. All went well for four days. Then symptoms of uræmia set in. The blood-urea rose to 200 mgm. per 100 c.c., and a week after the operation the patient died.

Fortunately the renal condition can be improved by regular drainage of the bladder. The deficiency in the renal function is usually due not so much to atrophy of the renal parenchyma from back pressure, as to disturbances in the circulation of the kidneys, as shown by Kornitzer, Hineman and Morrison. This is why one often sees a rapid improvement in the renal function as soon as the bladder is regularly emptied by a catheter.

The following is a brief report of a case under personal observation:—

Patient, aged 67, admitted to hospital with a greatly distended bladder and in a state of deep uræmic coma with convulsions; blood-urea of 260 mgm. per 100 c.c., blood-pressure 180. Infusions of dextrose solution and gradual decompression of the over-distended bladder restored the patient to consciousness and slowly improved his condition. The blood-urea fell from 260 mgm. to 140 mgm. in three weeks. A fortnight later it was 73 mgm., and finally it fell to normal. At the same time the condition as shown by other tests of renal function improved so much that prostatectomy became possible after five months' pre-operative treatment. The patient recovered quickly and never showed any sign of uræmia after the operation, and is to-day, two years after the operation, quite healthy and able to earn his living as a workman.

Suitable pre-operative treatment usually results in re-establishing the renal function and in rendering the patient fit for prostatectomy. There are, however, a few patients whose kidney function cannot be improved sufficiently by catheter drainage. They should not be submitted to prostatectomy, but a suprapubic fistula should be established.

Occasionally we see patients who do not give quite so bad a response to the kidney tests as to render radical operation impossible, but in spite of drainage of the bladder do not improve sufficiently to enable us to perform prostatectomy without any fear of uræmia. In such cases one is rather inclined to risk an operation if the general health of the patient is good. But if the function of the heart and lungs is not quite satisfactory, we should refuse operation, as slight vascular trouble may easily reduce the already impaired renal function to such a degree that uræmia sets in.

In deciding whether to operate or not, it is most important to ascertain if a perineal prostatectomy can be performed instead of a suprapubic. My opinion is that the perineal operation puts much less strain on the heart and lungs than the suprapubic, and it gives rise to much less shock than the suprapubic operation. I have found that suprapubic prostatectomy is usually followed by an increase in the blood-urea, lasting for several days, while after a perineal operation there is practically no increase in the quantity of urea in the blood. More than 50 per cent. of my patients submitted to suprapubic prostatectomy showed an increase in the amount of blood-urea on the fourth and fifth day after operation. In many of them it only amounted to 20 mgm. to 30 mgm. in 100 c.c. of blood, but in the majority of them it amounted to 60 mgm. to 70 mgm., and in one patient to more than 100 mgm. In only 18 per cent. of patients operated on by the perineal method was there a trifling increase in the blood-urea. In the majority of cases it was unchanged by the operation, while in 28 per cent. of them the blood-urea was less on the fourth or fifth day after operation than it had been before. This difference may be explained by the fact that the general vascular circulation is much less disturbed by the perineal than by the suprapubic operation. The perineal operation does not cause nearly so much bleeding, and the perineal wound does not hinder respiration or expectoration. Further, the perineal operation is not followed by necrosis of the tissues and the wound is well drained, so that there is only a slight amount of absorption which may increase the blood-urea.

54 Wildbolz: *The Indication and Execution of Prostatectomy*

MY TECHNIQUE OF PERINEAL PROSTATECTOMY.

The incision is the same as that employed by Zuckerkandl, Albarran and Proust, and, with a slight modification, also by Young. The central tendon of the perinæum is exposed and cleaned. A small transverse incision is made in it close to the bulb until the recto-urethralis muscle is exposed. It is divided closed to the urethra, and the prostate is exposed by blunt dissection. On the anterior wall of the wound are the bulb and musculus transversus perinei, while the rectum lies on its posterior. The prostate is pressed down towards the perinæum by means of a metal catheter introduced into the urethra. It is covered by the fascia of Dénonvillier. This fascia is divided transversely just above the apex of the gland and is pushed backwards, exposing the posterior portion of the capsule in the wound. I make a vertical incision in the middle line, commencing at least 1 cm. above the apex. It only involves the capsule of the prostate and does not penetrate to the urethra, and it enables us to distinguish between the capsule and the adenomatous masses. Both lobes of the prostate are enucleated as far as possible. The prostatic urethra is divided transversely just at the lower end of the adenomatous mass, the upper end being still left in connexion with the neck of the bladder. Young's retractor is introduced through the prostatic urethra into the bladder and the upper end of the urethra is divided close to the neck of the bladder, with any adhesions still existing between the adenoma and the capsule of the gland. The bladder is washed out to promote the removal of blood-clots.

Four sutures are placed through the neck of the bladder and afterwards through the stump of the urethra at the apex of the gland. They unite the neck of the bladder to the urethra inside the prostatic cavity, and, as far as possible, restore the normal anatomical conditions. Before the sutures are so tied the metal catheter is replaced by a silk one which is retained for twelve to fourteen days after the operation. A drainage tube is placed in the opening of the prostatic capsule, which is partially closed by means of a single suture. No packing is inserted. The superficial part of the wound is brought together by a few sutures. As a rule the wound heals per primam. The perineal drainage tube is removed in from three or four days after operation, and the fistula heals so quickly that the catheter can be removed in from ten to fourteen days. The patient is then able to pass water normally through the urethra.

The principal points on which this method differs from other perineal operations are the following:—

(1) Complete resection of the part of the prostatic urethra encircled by the adenoma. The verumontanum is usually uninjured.

(2) Suture of the neck of the bladder to the urethral stump. This suture not only protects the wound from contamination by urine but it controls bleeding from the neck of the bladder.

(3) Suture of the incised prostatic capsule.

This method of perineal prostatectomy does very little damage to the tissues. Loss of blood is, as a rule, trifling, and serious post-operative hæmorrhage rarely occurs. In only one case, in which the patient was badly infected, was there a serious post-operative hæmorrhage which necessitated opening the bladder and packing its neck with gauze on the eighth day. In two other cases I was obliged to pack the perineal wound three days after operation in order to stop the hæmorrhage which followed the removal of the perineal tube.

Symptoms of infection are extremely rare after perineal prostatectomy. I have only seen them in four patients out of more than 300, the reason being that the wound is smooth, has no cavities and no flaps of necrotic tissue, and is, in addition, drained ideally.

In cases of cancer of the prostate, symptoms of sepsis are more frequent, especially when the whole of the growth cannot be removed.

The perineal operation restores the anatomical condition of the bladder neck and prostatic urethra almost to normal, so that the bladder is completely emptied by spontaneous micturition, and in almost every case infection soon subsides.

The mortality of the perineal method is much lower than that of suprapubic prostatectomy. In 105 suprapubic operations I had a mortality of 15 per cent. In 305 perineal operations the mortality was 6·5 per cent. I doubt whether we shall be able to reduce it much below 5 to 6 per cent. as there is always a risk of pneumonia or cardiac failure after operation. The majority of my patients were healed in two or three weeks after perineal prostatectomy. In only twenty-nine cases was the healing of the perineal fistula delayed. In twenty-two of them it took from four to five weeks, and in seven from five to eight weeks for the fistula to heal. In only one case the fistula persisted for more than three months.

The greatest disadvantage of the perineal operation is the risk of wounding the rectum. In seven cases the rectum was wounded. In six of them the accident occurred among the first hundred patients operated upon, and in only one case in the succeeding two hundred. In three cases there was a persistent urethro-rectal fistula which required another operation for its relief. In three cases the wound of the rectum healed spontaneously. One patient who had been treated with large doses of X-rays had a small fæcal fistula in the perinæum following secondary necrosis of the tissues three months after prostatectomy.

Another risk of perineal prostatectomy is that the patient may show signs of incontinence after operation. It occasionally happens that there may be slight weakness of the bladder for the first four or five days after removal of the catheter. A few drops of urine may leak when the patient is walking, laughing, coughing, etc., but after a few days he is able to retain the urine well. Partial incontinence lasting for a few weeks was observed in only six cases, and in five others it persisted for more than four months. The incontinence was never complete, and was only observed on contraction of the abdominal muscles.

The advantages of the perineal operation are:—

(1) The operation is performed under the sense of sight.

(2) It is therefore possible to secure a good hæmostasis and to restore the natural anatomical conditions at the neck of the bladder.

(3) There is little damage to the tissues.

(4) Drainage is excellent.

(5) Breathing and coughing are unrestricted after the operation.

(6) There is a lower mortality than by the suprapubic method.

I only make use of the suprapubic operation when the pelvis is extremely narrow or when a combination of prostatic adenoma with papilloma or diverticula contra-indicates the perineal route.

[*June* 23, 1927.

The Stump of the Ureter after Nephrectomy—The Indications for Primary Nephro-ureterectomy.

By Frank Kidd, M.Ch., F.R.C.S.

[ABSTRACT.]

Nephrectomy, first performed by Simon in 1869, remained a dangerous operation until the ureteric catheter and the tests of renal function robbed the operation of its high mortality.

Intensive study of the renal blood-vessels further diminished the risks by warding off the dangers of hæmorrhage after operations on the kidney. Researches on the perirenal fat put in the hands of urologists a method of extracapsular nephrectomy whereby improved results,

56 Kidd: *The Stump of the Ureter after Nephrectomy*

as regards freedom from recurrence, could be achieved after nephrectomy for malignant disease, and in cases of tuberculous kidney made it possible to obtain first intention healing in an increasing number of such cases. The development of pyelography, ureterography and cystography rendered available a complete anatomical and pathological study of both sides of the urinary tract, so that except in cases of emergency the surgeon is no longer justified in embarking on nephrectomy until he has made such investigations, not only of the side suspected of disease, but also of the side supposed to be healthy. Such studies should be designed to exhibit not only the state of the renal pelvis but also the exact state of the whole length of the ureter from the kidney to the bladder. It is the purpose of this paper to suggest that painstaking attention should be paid to the condition of the ureter before embarking on nephrectomy, as in selected cases the removal of the larger portion of the ureter in addition to the kidney, will be found to give very much better after-results than nephrectomy alone.

Progress in the safety of nephrectomy in the hands of professed urologists may be said to have reached its limits, but progress in the prevention of complications caused by the stump of the ureter left behind after nephrectomy, has not yet received the attention it deserves. More and more, as time goes on, the urologist will find it necessary to remove a large part, if not the whole, of the ureter at the same time as he carries out nephrectomy. Many poor results obtained in the past after nephrectomy can be traced to a failure to realize the importance of removing a sufficient portion of the ureter.

It is surprising how little attention has been paid to the fate of the lower end of the ureter left after nephrectomy. This is probably because the stump of the ureter has not given trouble sufficiently often to any one individual to induce him to investigate the problem. When however any urologist reflects without prejudice on his past results he will probably recall that in more cases than he cares to remember, the stump of the ureter has given trouble after nephrectomy; but that the trouble has either remedied itself with time or with the adoption of palliative treatment, or has been glossed over. In times past when surgeons were accustomed to use silk for ligaturing the ureter it was not unusual for a stone to form round the silk ligature; and the stone was either passed with an attack of colic or had to be removed at a second operation. Even since the practice of using silk for ligature of the ureter has been abandoned, it has happened that some weeks after an operation for nephrectomy, especially on an infected kidney, the patient has suddenly been seized with an attack of fever, renal colic on the side of the nephrectomy, and strangury. The attack has been relieved by the passage of a quantity of pus, which has included portions of the catgut ligature.

> For instance, I operated upon a woman in December, 1925, and removed a foul calculous pyonephrosis in the right side, the ureter being burnt across and tied off with catgut, and the wound closed without drainage. The left kidney was healthy. On the eleventh evening the patient developed a right renal colic, followed by strangury and high fever, lasting for three days. On the fourteenth evening the sudden passage of clotted pus containing the catgut ligature led to the immediate relief of all symptoms. In July, 1926, she was found to have had no further trouble, and the urine was clear and sterile.

In cases of hydronephrosis, if the obstruction to the ureter is low down in the pelvic ureter and the ureter as far as the obstruction is not removed, the lower end of the ureter left behind more often than not gives rise to trouble. The patient may complain of repeated attacks of ureteric colic very similar to the attacks caused by the hydronephrosis, or the ligature may slip off and the patient be faced with a sinus continually discharging mucus or even urine. Both of these conditions will call for a secondary ureterectomy. Worse still, the stump of the ureter may remain infected and cause attacks of colic, fever and strangury. Such attacks may yield to regular lavage of the stump through a ureteric catheter. But in badly infected cases with severe obstruction at the lower end of the stump a large infected pouch may form, as Hyman has shown.[1]

> For example, I performed an emergency operation in the middle of the night on a woman in the country in October, 1919. The patient was gravely ill, with high fever and a large tender left kidney, the right kidney being healthy. A very large infected hydronephrosis full of foul purulent urine caused by an aberrant tail artery was removed.

1 *Annals of Surgery*, September, 1923, 387.

The ureter below the artery was not dilated, yet must have been infected. The patient was seen again in February, 1920. She had continued to suffer pain similar to her old left renal pain, but it was not so severe, and was accompanied by attacks of strangury, pyuria and fever. By means of the ureteric catheter the left ureteric stump was found to be infected, but not stenosed. The right kidney was healthy. The stump was washed out several times with colloid silver through a ureteric catheter, and the symptoms cleared up completely.

In 1921 I examined a woman, aged 66, since 1910 had suffered from attacks of strangury, fever, left renal pain, and profound wasting. The left kidney was removed. It contained a stone in one of the upper calyces. The kidney substance was thinned out, so also was the pelvis and the whole of the ureter, which was dilated to the size of an index finger. The ureter was cut across high up in the loin and left behind. She was not seen again until March, 1927. She states that she has been much better in herself, but has attacks of left ureteric colic, accompanied by strangury and fever, which are relieved by the passage of purulent urine, after which the urine remains clear for many months. The urine contained a moderate degree of pus, and the right kidney was healthy. It was found impossible to pass a No. 5 ureteric catheter up the left ureter. There was undoubtedly a narrow stricture of the lower end of the left ureter, and the attacks were due to occasional retention of purulent secretion in the ureteric stump.

The contents of an infected and stenosed ureteric stump may leak into the surrounding tissues causing a dense, fibro-fatty mass of tissue containing loculated masses of pus and even urine, occasioning such severe symptoms as to render the life of the patient a misery, unless a secondary ureterectomy—an operation which may present formidable difficulties—is carried out. The cases quoted below illustrate how these complications may be prevented by primary nephro-ureterectomy, an operation which presents far less difficulty than a secondary ureterectomy, as it is carried out before the onset of these late inflammatory changes.

After nephrectomy for tuberculosis of the kidney, on rare occasions the catgut ligature may slip, giving rise to a urinary fistula in the kidney wound which calls for a secondary ureterectomy. I have met with two such cases. More frequently a pyo-ureter develops and bursts, in the course of five or six weeks, into the peri-renal tissue giving rise to an abscess in the nephrectomy wound. This leaves a tuberculous sinus which may refuse to heal till many months have elapsed. Such complications are to be expected if the tuberculous ureter is grossly thickened, and especially if fibrosis sets up definite obstruction to the exit of the contents of the infected ureter into the bladder. For these reasons Lilienthal[1] has advocated nephro-ureterectomy. Some surgeons, notably Mr. Nitch and Mr. Jocelyn Swan, in this country, are accustomed, in all cases of tuberculosis of the ureter, to remove as large a portion as possible of the lumbar and pelvic ureter. Others, such as Hugh Young and Rovsing, consider that this step entails risk of the formation of an abscess or urinary fistula deep in the pelvis and hold that it is safer to free the lumbar and pelvic ureter from the kidney and to bring it out through a small muscle-splitting incision in the inguinal region, so that it can drain on to the surface of the skin. Which is the safer method is a subject ripe for discussion. Probably all will agree that there is no need for ureterectomy in those cases in which the tuberculous ureter is merely glassy with submucous tubercle and is not grossly thickened and stenosed through peri-ureteric tuberculosis.

In cases of septic stone impacted for many years in the pelvic ureter, it is not always sufficient to remain content with a mere removal of the stone. If the ureteric catheter reveals that the kidney and ureter above the stone are mere dilated, atrophic-septic or aseptic-relics, it is probably far wiser to make a clean sweep by means of primary nephro-ureterectomy.

Raymond Latchem, in a paper from the Mayo Clinic,[2] published the following conclusions at which he had arrived from his experimental studies on animals:—

(1) In the normal ureter after nephrectomy no attempt is made toward obliteration of the lumen by disappearance or atrophy of the mucous membrane, but there is a noticeable atrophy of the muscular coat.

[1] *Annals of Surgery*, April, 1911.
[2] *Journal of Urology*, 1922, viii, 257.

58 Kidd: *The Stump of the Ureter after Nephrectomy*

(2) In the hypertrophic hydro-ureter or pyo-ureter, with drainage of its contents after nephrectomy, the mucous membrane remains intact and the muscular coat gradually atrophies.

(3) In the hypertrophic hydro-ureter or pyo-ureter with complete obstruction to drainage of the ureteral contents after nephrectomy, the mucous membrane remains intact and the muscular coat remains hypertrophic.

(4) Absorption of the contents of a distended ureter is very limited if it occurs at all.

(5) If infection is present in the contents of the ureter, it may spread through the wall and give rise to peri-ureteral infection and abscess formation.

It seems clear, then, from both experimental and clinical evidence, that if there is no obstruction to the stump of the ureter after nephrectomy between the ligature and the bladder, the mucous membrane will not disappear, but the muscle wall will atrophy to a certain extent and ureteric peristalsis will still continue in a mild degree to extrude from time to time a small quantity of mucus secreted by the wall of the ureter. If there is disease of the wall of the ureter, as for instance, infection after hydronephrosis, pyonephrosis or tuberculosis and yet no obstruction to the lumen of the ureter, complications will seldom arise, the inflamed and infected ureter will gradually atrophy and its contents become sterile. If the process of sterilization be delayed it can be hastened by washing out the stump of the ureter with a ureteric catheter. The author has actually observed atrophy and healing of the tuberculous ureter exposed during abdominal operations for other diseases, in cases in which nephrectomy for tuberculous kidneys had been carried out some years previously; and he has noted that in most cases after removal of a stone pyonephrosis the stump of the ureter has become aseptic and has given no further trouble. On the other hand, if there is any real degree of stricture between the site of the ligature on the ureter stump and the entrance of the ureter into the bladder, the result of congenital, inflammatory or calculous stricture, then trouble is likely to arise in the stump of the ureter, as the muscular wall will hypertrophy in an endeavour to get rid of the contents of the ureter, causing attacks of ureteric colic. If the contents of the ureter are infected the lumen of the ureter will become dilated so as to form an infected pouch, causing attacks of colic, fever and pyuria. In severe cases the infection may spread through the wall of the ureter, setting up dense fibro-fatty adhesions and even peri-ureteric abscesses.

Far greater care, therefore, should be exercised in taking not only pyelograms but ureterograms of all cases in which nephrectomy is about to be carried out. If these show the slightest degree of stricture at any point in the pelvic ureter, then not only should nephrectomy be carried out but the stump of the ureter should be removed until a point is found above the bladder where the ureter is normal and not stenosed. If the ureter is ligated and cut off here no trouble will arise symptomatically after the operation, and in cases of tuberculosis it is in the highest degree unlikely that any infection of the pelvic tissues or the formation of a urinary fistula will occur.

In cases that arise commonly the indications for primary nephro-ureterectomy are as follows:—

(1) In hydronephrosis, simple or infected, where the stricture is low down in the pelvic ureter.

(2) In pyonephrosis or atrophic hollowed septic or aseptic kidney secondary to a stone long impacted in the pelvic ureter.

(3) In tuberculous pyonephrosis associated with inflammatory stricture in the pelvic ureter.

(4) In all other types of pyonephrosis associated with inflammatory stricture in the pelvic ureter.

Rare conditions in which primary nephro-ureterectomy is indicated are the following:—

(1) Papilloma of the renal pelvis with secondary deposits in the ureter.

(2) Primary carcinoma of the ureter.

(3) Congenital opening of the ureter into the wall of the vagina, associated with congenital cystic or atrophic kidney.

(4) Ectopic pelvic kidney.

(5) Heminephrectomy for horse-shoe kidney associated with infected stone.

(6) Primary fibro-fatty ureteritis, a rare condition in which infection has extensively damaged the wall of the ureter yet has left the kidney comparatively free.

The operation of primary nephro-ureterectomy can be performed in many different ways. In the majority of cases I cut down on the kidney first from behind, and after the kidney has been freed and its pedicle tied, I free the lumbar ureter from its connexions, taking care to tie all bleeding points, which may be numerous. The ureter can be burnt across with a cautery, or the kidney and ureter be left hanging out of the lower margin of the wound, which is then stitched up and the patient turned on to his back. In the easier cases when the obstruction, such as a stone, is high up in the pelvic ureter, the ureter is then exposed through my muscle-splitting incision,[1] tied off with catgut below the point of obstruction and burnt across, and the ureter, with the kidney, is drawn out from the wound in the back. If a drain is carried to the stump of the pelvic ureter this drain should consist of a piece of rubber glove. Tubes endanger the wall of the iliac vessels and should not be employed. If on the other hand the case is a difficult one, in which the obstruction lies close to or even in the walls of the bladder, then a better exposure can be obtained by turning the rectus muscle outwards from near the middle line below the umbilicus. Through this incision the ureter can be stripped extraperitoneally to the wall of the bladder, the numerous branches of the internal iliac and other vessels running to the wall of the pelvic ureter and bladder can be exposed and tied off, and the ureter be completely removed. In some cases it has even been found necessary to remove a portion of the bladder wall, a step which is perfectly feasible, through this incision. Drainage, if considered necessary, can be carried out through a small puncture counter-opening in the lateral abdominal wall. In women the uterine artery can be avoided by exposing the ureter in front of the broad ligament and tying it off close to the bladder, after which the ureter can be freed higher up in the pelvis behind the broad ligament, and the ureter pulled upwards and backwards through the broad ligament. The operation may be varied in certain cases by cutting down on the pelvic ureter first. After it has been dealt with and freed the kidney can then be exposed through a second incision, and the whole specimen removed.

The operation is usually more easily performed in this way, but in certain cases the surgeon will probably wish to expose the kidney first and satisfy himself as to its exact condition before removing it. The operation can be carried out through a long sloping incision carried from the back of the loin round the lateral abdominal wall to the edge of the rectus muscle—an operation much favoured by Professor Israel in the past. Two separate incisions are less mutilating and are preferred to this single long incision.

[Twelve operation specimens illustrating most of the above conditions were exhibited and will be fully described elsewhere.]

[1] *Lancet*, June 7, 1913.